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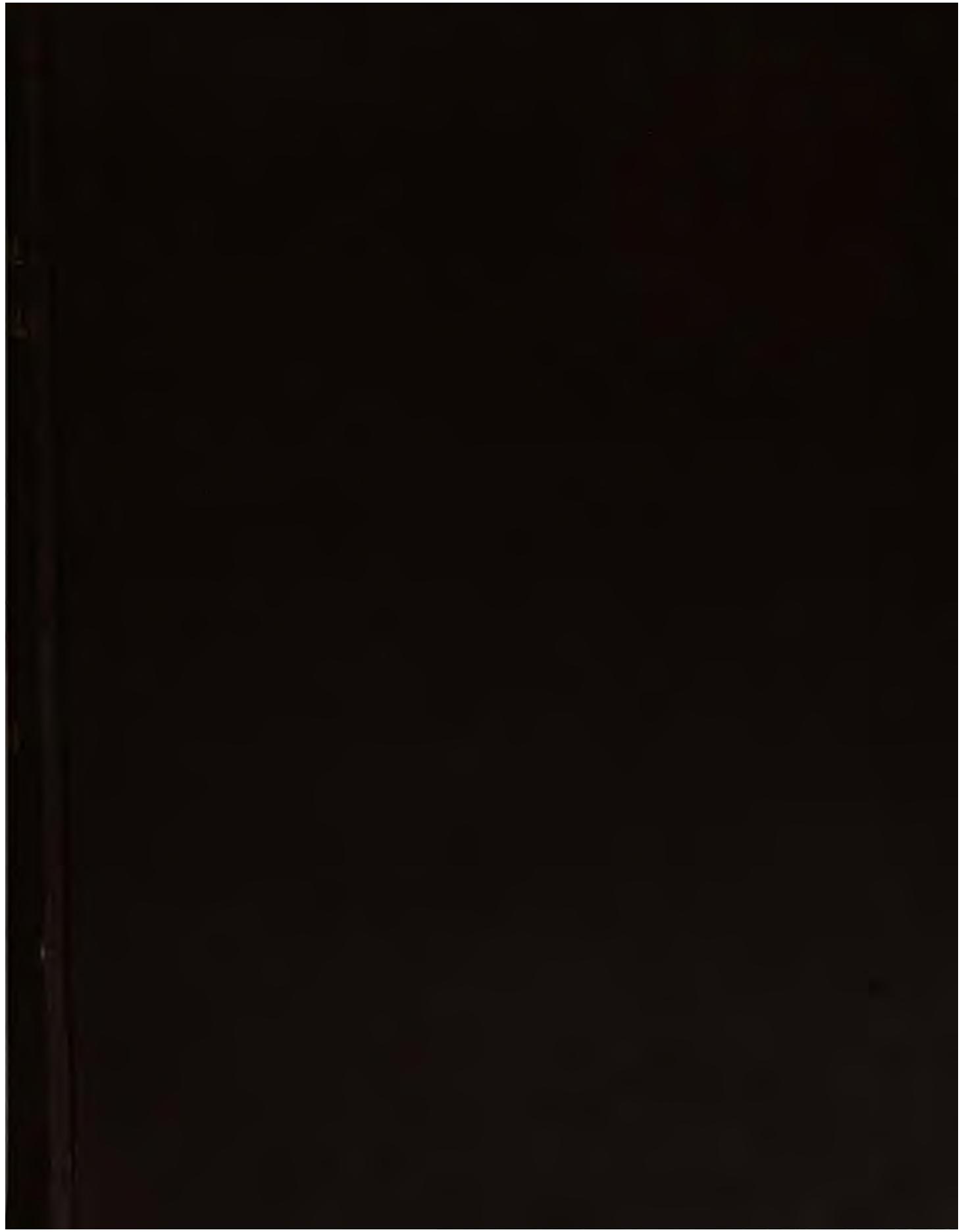
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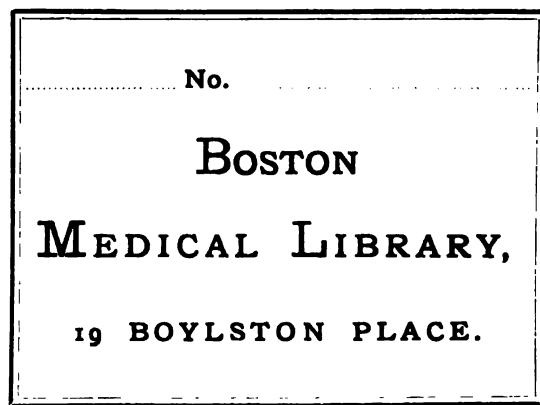
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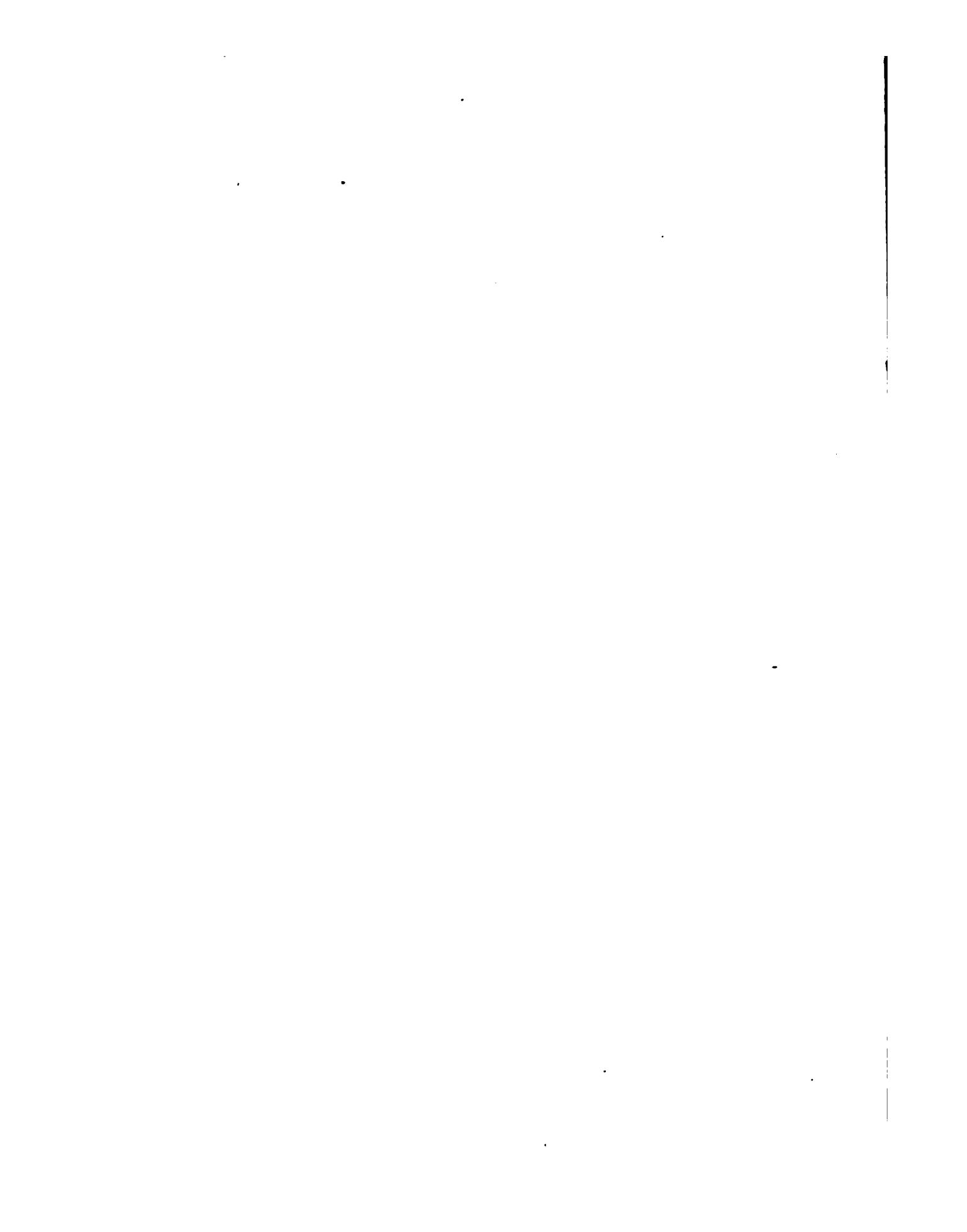
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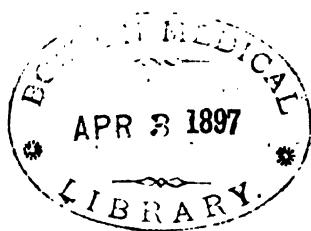
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LIST OF CONTRIBUTORS.

	PAGE
BARKER, ARTHUR E., F.R.C.S. Weak Scars and Herniæ following Abdominal Sections	193
BEEVOR, C. E., M.D., F.R.C.P. Paralysis of the Soft Palate 376
BIDWELL, LEONARD A., F.R.C.S. Some Methods of performing Intestinal Anastomosis	... 337
BISS, C. Y., M.D., F.R.C.P. Intra-thoracic Aneurysm 136
BOYD, M. A., M.D. Defective Metabolism in Relation to Gout 197
BRADFORD, J. ROSE, M.D., F.R.S. Notes from the Clinic 232
On a Case of Muscular Atrophy 294
BRYANT, THOS., F.R.C.S.Eng., M.Ch. Clinical Notes 373
BUTLIN, H. TRENTHAM, D.C.L., F.R.C.S. Clinical Notes 89
CARR, J. WALTER, M.D., M.R.C.P. Pneumonias in Children and their Sequelæ 241
CAVAFY, JOHN, M.D., F.R.C.P. Enteric Fever 221
CLARK, ANDREW, F.R.C.S. A Note from the Clinic 302
COLLINS, E. TREACHER, F.R.C.S. Orbital and Ocular Tumours 5
COWELL, GEORGE, F.R.C.S. Iridectomy 92
DIEULAFOY, Prof. A Study of Appendicitis 310, 327
DUCKWORTH, SIR DYCE, M.D., LL.D. Clinical Notes 17
GARDNER, H. B., M.R.C.S. Administration of Nitrous Oxide Gas, with Oxygen, for Removal of Adenoid Growths 301
GOULD, A. PEARCE, M.S., F.R.C.S., M.B., L.R.C.P. Notes from the Clinic 257
Cancer of the Rectum 356
GOWERS, W. R., M.D., F.R.C.P., F.R.S. Slight Multiple Neuritis 21
GREEN, T. HENRY, M.D., F.R.C.P. Chronic Bright's Disease 273
GUTHRIE, LEONARD G., M.A., M.D., M.R.C.P. Unilateral Paralysis of the Ocular Sympathetic	... 225
HABERSHON, G. H., M.D., F.R.C.P. Mitral Stenosis 129
HADLEY, WILFRED J., M.D., M.R.C.P., F.R.C.S.Eng., Cerebral Tumour, and a Case of Arsenical Poisoning 369
HARRISON, REGINALD, F.R.C.S. Stricture of Urethra 204
HAWTHORNE, C. O., M.B. Cancer of Liver 361
HEATH, CHRISTOPHER, F.R.C.S. Tetanus 177
HOPKINS, JOHN, F.R.C.S. Surgical Sequelæ of Chronic Nerve Disease 113
HUTCHINSON, JONATHAN, F.R.S., LL.D. Clinical Cases	... 24, 206, 230, 277, 333, 353
JACOBSON, W. H. A., M.A., M.Ch., F.R.C.S., Epithelioma of Tongue 87
JOHNSON, G. JAMESON, M.A., M.B. The Application of Hernial Trusses 299
JOHNSON, RAYMOND, M.B., B.S., F.R.C.S. On Hernia in Childhood 321
LANE, W. ARBUTHNOT, M.B. Lond., F.R.C.S. Clinical Notes 10
Notes in the Out-patient Room 151
LEES, DAVID, M.A., M.D., F.R.C.P., M.R.C.S. Appendicitis 17

	PAGE
LOCKWOOD, C. B., F.R.C.S. An Unsuitable Case of Hernia for Radical Cure 134
A Suitable Case for Operation of Radical Cure of Hernia 401
LUFF, A. P., M.D., B.Sc. Clinical Notes 59
MACCORMAC, SIR WILLIAM, M.A., D.Sc., F.R.C.S. Some Causes of Haematuria 65
Internal Derangement of the Knee-joint and Loose Cartilages in Joints 209
Cancer of the Tongue 289
Diseases of the Tongue 385
MACKENZIE, STEPHEN, M.D., F.R.C.P. Diagnosis of Cirrhosis of Liver 81
McKERRON, R. G., M.A., M.B. Accidental Haemorrhage 28
MARTIN, SIDNEY, M.D., F.R.S., F.R.C.P. Treatment of Diphtheria by Antitoxin Serum, Lecture II. 1
MORTON, CHAS. A., F.R.C.S. The Surgical Treatment of Tuberculous Disease of the Bladder 157
ORMEROD, J. A., M.A., M.D., F.R.C.P. Friedreich's Disease 118
PICK, T. PICKERING, F.R.C.S. Operative Treatment of Diseases of Hip-joint in Children 161
POLLARD, BILTON, B.S., F.R.C.S. Ectopia Testis 97
Congenital Tumour of Sterno-mastoid Muscle 218
POWER, D'ARCY, M.A., F.R.C.S. Meningitis in its Surgical Aspects 49
ROLLESTON, H. D., M.A., M.D., F.R.C.P. Clinical Notes 141
Cirrhosis of the Liver in Children 305
ROUTH, AMAND, M.D., B.S., M.R.C.P. Procidentia Uteri 145
SHEILD, A. MARMADUKE, M.B., F.R.C.S. Notes from the Clinic 389
SMITH, F. J., B.A., M.D., F.R.C.S., F.R.C.P. Clinical Notes 188
SPICER, W. T. HOLMES, M.B., F.R.C.S. Lymphatic Conjunctivitis 262
STEWART, WM. R. H., F.R.C.S., L.R.C.P., &c. Examination of the Ear 123
Some Complications of Middle Ear Suppuration 340
STOKER, GEO., M.R.C.P.I. Impaired Movements of the Vocal Cords 104
Chronic Glandular Disease of the Nose and Naso-pharynx 403
TAYLOR, FREDERICK, M.D. An Unusual Case of Hodgkin's Disease 33
TAYLOR, SEYMOUR, M.D., M.R.C.P. Treatment of some Medical Emergencies 41
TOOTH, H. H., M.A., M.D., F.R.C.P. Clinical Notes 100
TURNER, J. G., F.R.C.S. Antral Suppuration following Invasion by a Dental Cyst 394
WATERHOUSE, HERBERT F., M.D., C.M. EDIN., F.R.C.S. Eng. Adenoid Vegetations in the Naso-pharynx, and their Treatment 276
WETHERED, F. J., M.D., F.R.C.P. Types of Pulmonary Tuberculosis 168
WHISTLER, W. MACNEILL, M.D., M.R.C.P. Syphilis as it affects the Larynx 180
WILKIN, G. C., M.R.C.S. Polyp of Right Frontal Sinus 28
WILLIAMS, C. THEODORE, M.A., M.D., F.R.C.P. Arrest of Pulmonary Tuberculosis 74

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TWO LECTURES

ON THE

TREATMENT OF DIPHTHERIA BY ANTITOXIC SERUM.

LECTURE II.

Delivered at University College Hospital.

BY

SIDNEY MARTIN, M.D., F.R.S., F.R.C.P.,

Assistant-Physician to the Hospital.

GENTLEMEN,—In the first of these lectures I went over some of the scientific points in regard to the properties of antitoxin. To-day I want to go over the clinical aspects of its use. The investigation of the toxin and antitoxin has brought forward remarkable facts, and instituted a new era in pathology; but as practical men we have to deal with the effects of antitoxin on diphtheria, and with what it actually does. Is it useful in aiding the cure of diphtheria? Before going on to discuss the cases that have been treated here, we must first discuss one or two points with regard to the bacterial cultivation. Seventy-five cases have been treated in this hospital from December, 1894, to December, 1895, and cultivations were made in seventy cases. Sterilized cotton wool on a rod was rubbed over the throat, and then smeared over the surface of solidified blood serum. The tube is placed in the incubator for sixteen or twenty-four hours, at the end of which time there is a surface growth. It is in the majority of instances a mixed growth.

The bacillus diphtheriae was found in sixty-five out of the seventy cases, which is a greater proportion than that reported by Dr. Roux, who states that in 20 or 30 per cent. of the cases the bacillus was not found. The bacillus was found mixed with cocci, and in two cases with an excess

of streptococcus, the association of which, with the bacillus in the throat, must be considered of grave significance.

Now, as to the five cases in which when examined for bacteria no diphtheria bacilli were found. Streptococcus only was found in one case. In three of the other four cases, streptococci and staphylococci were found. It was found that these cases were diphtheritic, because of the symptoms developed afterwards. You may examine the throat four or five times at different periods, and not find any bacillus diphtheriae, and yet that case may turn out to be a case of diphtheria. The diagnosis in these cases is made on the loss of knee-jerk, and on the development of squint, and of regurgitation of liquids. You cannot rely exclusively on the bacteriological examination. If you do not find the bacillus in one or even two examinations, you must not conclude it is not a case of diphtheria.

The next point is the mode of administration of the antitoxin. It may be administered in perfect safety only in one way—that is, with strict antiseptic precautions. The liquid is serum in which bacteria will readily grow, unless it is perfectly sterile. If you use a dirty syringe, you may inject infective matter in a liquid which is a good culture for bacteria. You may inject bacteria with it, and therefore get an abscess. Both syringe and needle must be boiled for ten minutes; and the skin should be well washed, first with soap and water, and then with 1 in 20 carbolic. The serum is injected in the loin or in the back; and as a rule, we do not inject more than 30 c.c. at one time. Another mode is one which has been used in this hospital, namely, the intravenous injection, and it has been very successful. It is practically reserved for tracheotomy cases, because in a child that is not under an anaesthetic the needle is not easily inserted into a vein. The best mode of procedure is, when the child is coming to from the anaesthetic after the tracheotomy, to put a ligature round the arm, and to make an incision an inch long over the median basilic vein; when the vein is exposed, the needle

is thrust into it, and the injection made in the direction of the blood stream.

No bleeding occurs if a fine needle be used. The wound is stitched up, and heals by first intention. No serious results follow, and the two cases in which it was performed did perfectly well. A little rise of temperature occurred in one case, but the child was perfectly well afterwards.

The bad effects which may be ascribed to the antitoxin are the occurrence of a rash, joint-pains, and swelling of the joints. Here are the facts as they occurred in the cases in this hospital. Seventy-five cases were injected with the antitoxin, from 10 c.c. to 80 c.c.: in thirty-two of these cases a rash followed; in one case a slight rash appeared on the third day, in twenty-two cases it appeared on the seventh to the tenth day, and in nine cases from ten to seventeen days; therefore, it comes out, as a rule, from the seventh to the tenth day. There may be two rashes, the first one which disappears in a few days, and then a second appearing after the fading of the first rash. This is a very important matter, because the rash has been mistaken for the rash of an acute specific disease. Eleven out of seventy-five cases were under five days in the hospital; there were thus sixty-four cases only in which there was the possibility of a rash being noted. Judging from these figures, the rash may be said to occur in about fifty per cent. of the cases. The rash is either an erythematous blush over the body, trunk, arms and legs, or it is red and papular, or urticarial. In the majority of cases it is an erythematous rash, the urticarial variety being less common. As a rule, it is not accompanied by any rise of temperature; in only five of the cases was there a rise in temperature noted. One patient had pain in one knee-joint, and insignificant swellings in both wrists. The effusion in the knee-joint caused some discomfort, but was relieved by local applications.

Sometimes the rash seems to begin at the point of the injection, but in the majority there is no relation to the site of injection. No abscess or inflammation occurred at the site of injection.

It has been stated that antitoxin tends to produce Bright's disease; but to any one who has seen many cases of diphtheria that statement is utterly fallacious; nephritis is a rare complication of diphtheria. Anuria may just precede death,

with vomiting, and the appearance of these symptoms is a fatal sign in diphtheria, whether treated by antitoxin or not.

The beneficial effects of the antitoxic serum may now be discussed. There is no immediate result from the injection. Indeed, during the first twenty-four hours after the injection of antitoxin, the membrane sometimes spreads, which has led some hasty people to the conclusion that the antitoxin does not do any good. But if you go on watching the case you will find two things happen—the membrane separates very readily after a few days, and the general condition of the child is improved. The whole aspect of the disease changes after the injection of antitoxin, and all the children so treated are in a better bodily condition than those not so treated. One can say that the children, though they have come into the hospital with a poisoned look (they have that earthy pallor so characteristic of severe diphtheria), after a few days lose this pallor, showing that the antitoxin does act on the ill-effects of the disease. The effect on the membrane of the throat is definite. In thirty-one of the pharyngeal cases in which the record of the membrane is quite clear, the membrane disappeared in two to six days after the injection in seventeen cases; in eight cases in seven to ten days; in four cases in eleven to fifteen days; in two cases the membrane persisted for twenty-three days. The throat was clear then in ten days in the majority of instances. With the laryngeal cases one judges of the disappearance of the membrane from the time of the tracheotomy to that of the removal of the tube. In the twenty cases of successful tracheotomy the tube was removed, in six to twenty days in four-fifths of the cases, and the children were practically convalescent.

Now, what we have to do is to compare the effects of the antitoxin treatment during the last year (1895) with the treatment which has prevailed in the previous years. The antitoxin treatment preserved more lives than the other treatment previously adopted, which was usually the employment of antisepsics to the membrane, corrosive sublimate, carbolic acid, &c. In 1895 we injected the antitoxin, and treated the throat with a warm spray of bicarbonate of soda (twenty grains to the ounce), alternatively every four hours with a spray of corrosive sublimate (1-2000).

The following table shows the total mortality in the antitoxin year (1895) with that in each of the four previous years in which antitoxin was not used :—

LARYNGEAL.		PHARYNGEAL.		Total Mortality %
No. of Cases.	Deaths.	No. of Cases.	Deaths.	
1891 ... 30	21	32	6	41·9
1892 ... 25	15	35	5	33·3
1893 ... 41	32	64	7	37
1894 ... 34	16	30	9	39
1895 ... 30	10	45	11	28

Therefore, the total mortality is lower in the antitoxin year than in the best of the previous four years. The reduction in total mortality is clear, but, it may be asked, why is the mortality not less than that? Diphtheria is a disease which is more fatal to children under 5 years of age than over 5. The mortality in children under 5 is over fifty per cent. and in some years attains even seventy or seventy-five per cent. In 1891, of all children admitted under 5, there were 41 cases and twenty-two deaths (53·6 per cent. mortality), thirty-three cases and fifteen deaths in 1892 (45·4 per cent.), fifty-eight cases and thirty deaths in 1893 (51·7 per cent.), thirty-four cases and eighteen deaths in 1894 (53 per cent.), forty-eight cases and seventeen deaths in 1895 (35·4 per cent.). There is a great improvement in the mortality in the year 1895, and this is more marked in laryngeal cases, because the percentage of deaths in laryngeal cases has varied from fifty to seventy-five, whereas last year it was thirty-three. That is only part of the subject. One point is that the longer a diphtheria patient is untreated, the more toxin he has in his body, and, therefore, the more toxin you have to counteract. The fatty degeneration of the heart, and the degeneration of the nerves may be already present when the patient comes under treatment. What is the effect of antitoxin in those cases which are admitted in the first or early stage—on the first to the fourth day of the disease. Diphtheria is fatal to a child in ten or fifteen days, and later on death may result from the paralyzing effects of the poison. Speaking broadly, ten or fifteen days is the average duration

of a fatal case. The majority of cases are admitted into the hospital within the first four days, and the following figures apply to such cases. In 1891 there were thirty-nine cases and nineteen deaths, making forty-nine per cent.; in 1892, thirty-one cases and eleven deaths, making thirty-six per cent.; in 1893, fifty cases and fourteen deaths, making twenty-eight per cent.; in 1894, thirty-six cases and fourteen deaths, making thirty-nine per cent., and in 1895, there were forty-six cases and eight deaths, making seventeen per cent. That is the real point, when you begin the treatment in the first four days of the disease, you have an enormous reduction in the mortality. When the children are admitted from the fifth to the seventh day, or later, we get, in 1891, twenty-one cases and eight deaths, making 38 per cent.; in 1892, twenty-four cases and nine deaths, making 37 per cent.; in 1893, thirty-four cases and seventeen deaths, making 50 per cent.; in 1894, twenty-three cases and ten deaths, making 43 per cent.; and in 1895, twenty-eight cases and thirteen deaths, making 46 per cent. If the treatment is commenced late there is not much difference between the cases treated with the antitoxin and the old treatment. The mortality in cases admitted before the fourth day was only 17 per cent. in 1895. This mortality in 1895 is really better than is shown here, for the antitoxin is only a remedy for the diphtheria itself, and one has to consider what the causes of death were. One of the fatal pharyngeal cases died of tubercular meningitis, and not from diphtheria. One case died from streptococcus infection. One of the laryngeal cases died of empyema and pericarditis; the diphtheria had disappeared in this case, and the child died from secondary infection. Another died from chronic kidney disease. The other cases died from diphtheria; so that we have to exclude four cases. Diphtheria is one of the most fatal diseases we have to deal with, and yet, when treated with antitoxin, it has become one of slight mortality, provided the treatment is begun before the fourth day of the disease.

What is the effect on the paralysis which ensues? The degree of paralysis is due to the amount of toxin present. In severe cases there is more toxin than there is in a mild case, and the severer the case the earlier the paralysis ap-

pears. It is a practical point which you ought to lay to heart, because, as I said in my last lecture, one point by which we judge a severe case is the loss of knee-jerks, which is an early sign of paralysis. If you have a large amount of toxin in the body, and (I am putting a supposititious case) and that case is saved by the injection of antitoxin, you are more likely to get an increase in the number of cases of paralysis, owing to the severe cases having thus been saved from death and life prolonged. Provided a larger number are saved from death, the question of the resulting paralysis is, though serious, not hopeless, because in the majority of cases they get well from the paralysis. Fifty-seven of the cases were watched as regards paralysis, and were in hospital long enough for that purpose; and in 27 of the cases there were signs of the paralysis. In 15 the only sign was the loss of knee-jerks; in 12 there was as well paralysis of accommodation, squint, and regurgitation of liquids. All the cases recovered. One interesting point that has become evident is, that after the membrane had disappeared in cases treated by antitoxin, the bacillus persists, and this persistence is a very important point, because, if such a patient be allowed to mix with healthy people, infection is still possible. It is not simply that the bacillus persists for a day or two, but it may persist for weeks. In one case in this hospital—a child of 11 or 12—the bacillus was obtained in pure cultivation from the throat 35 days after the membrane had disappeared. The lad was in the hospital 57 days; the membrane disappeared in about 13 days, and the bacillus was present up to the 57th day. The child was quite well, walking about the ward, and you could find nothing abnormal, except a little redness of the throat. The bacillus *diphtheriae* finally disappeared after prolonged treatment locally with corrosive sublimate. In the ward now there is a child who has been in for a long time, and in whom the same difficulty in getting rid of the bacillus from the throat is experienced. It is a serious state of things, and one which we do not yet quite understand. The point is this—why should the bacillus remain in the throat without producing any local lesion? One child of 5 was admitted for laryngeal diphtheria, and had tracheotomy performed and antitoxin injected. The

child recovered, and remained in the hospital for thirty days. The tube was removed on the tenth day; the patient left the hospital well and with no paralysis. The child had been home a week when it was brought back by the mother with membrane in the throat again. In the membrane the bacillus was found, and laryngeal symptoms ensued, for which a second tracheotomy had to be done to the child through the old scar.

In the ward now there is a little child with pharyngeal diphtheria; the child apparently got well, and after a time membrane again developed; another dose of antitoxin has been given, and now the child is quite lively and well, and yet the bacillus is still in the throat. This may be explained by the fact that the antitoxin protects the tissues against the invasion of the bacillus, which means that the body is put into such a condition that the bacillus cannot grow in the tissues, and therefore cannot produce its poison. In this artificially produced immune condition, the patient can go about with bacilli in the throat without suffering any harm. This immunity which is produced by the antitoxin only lasts for a certain time. Directly the immunity diminishes then the bacillus again overcomes the natural resistance of tissues, and forms a membrane and produces symptoms. This is an extremely hopeful point of view, because, if we can produce this artificial immunity, we may hope to do a great deal in the treatment of this disease.

A final word may be said regarding the dose of antitoxin to be given. This, as I said in my first lecture, is not to be reckoned by the number of cubic centimetres, but by the number of normal units the serum contains; and all specimens of serum ought to have the number of units stated on the bottle. The stronger the serum the more efficacious it is; and it is best to inject the total dose for the case within 24 hours of the patient coming under treatment. This total dose ought not to be less than 4,000 normal units. It is important to inject the serum at the earliest possible opportunity; even if the case is not diphtheria, you do not do any harm to the child. I feel certain that an early injection of the serum in all doubtful cases will be the future treatment. If the rash comes out, what does the rash matter against the life of the child? But if you inject the serum early enough,

you put the child under the very best conditions of recovery. You produce in the child's body an artificial immunity against the disease, and you counteract in an early stage the action of the poisons that are formed in the body.

A POST-GRADUATE LECTURE ON ORBITAL AND OCULAR TUMOURS.

Delivered at the Royal London Ophthalmic Hospital,
Moorfields, March 25th, 1896.

BY
E. TREACHER COLLINS, F.R.C.S. Eng.,
Assistant-Surgeon to the Hospital.

LADIES AND GENTLEMEN,—The orbit contains a number of structures of such widely different nature, that the characters of the tumours arising in them are very various. The subject divides itself into :—

- (a) Tumours starting around the eyeball and optic nerve.
- (b) Tumours starting in the eyeball itself or optic nerve.

We will deal with tumours starting in the structures around the eyeball first. These are more strictly orbital tumours than those belonging to our second division, and they may be divided into

- (a) Cystic growths.
- (b) Solid growths.

The cystic are either dermoids or hydatids. Dermoid cysts are more frequently met with at the angle of the orbit than deeply in the orbit itself. The most common situation of them is at the outer and upper angle of the orbit, beneath the eyebrow. They are congenital cysts, and tend to increase throughout life, sometimes taking on sudden and rapid enlargement. They are deeply situated between the orbicularis muscle and the bone; on cutting down upon them it is often found that the bone beneath them is cupped. This cupping is not due to absorption of the bone from pressure of the growth, but to failure in its development in that position. The cysts being congenital, the bone grows around but not beneath them. I have only seen one case where a dermoid cyst was situated deep in the orbit itself. In this case, an

orbital tumour was diagnosed, and an exploratory incision was made with the object of finding out what was its nature. I examined the piece of tissue which was excised, the microscope showed it to be partly glandular and partly fibrous, and as the swelling was in the situation of the lachrymal gland I reported that the tumour was probably a fibro-adenoma of that structure. There still remained considerable swelling in the patient's orbit, and the surgeon proceeded to remove the rest of the growth. After he had cut down a little way, he came to a large cyst, from which sebaceous matter escaped. It was a dermoid cyst deeply placed in the orbit, which had pushed the lachrymal gland in front of it, and the piece which the surgeon removed at the first operation was the displaced lachrymal gland. It shows how easily a mistaken diagnosis may occur, even where a microscopical examination has been made. In removing dermoid cysts about the orbit the surgeon often has to cut very deeply, and after excision a comparatively large cavity is left. It is very important to thoroughly irrigate this cavity with antiseptic lotion, because if, as generally occurs, the cyst has been wounded in the process of its removal, some of the sebaceous matter which it contains will have escaped into the wound; and that, if left, will form a nidus for pathogenic germs. Before uniting the tissues after operation I usually put in a horse-hair drain.

The other form of cysts which occur in the orbit—the hydatid—may be situated beneath the conjunctiva or deep in the orbit. The symptoms of the latter are the same as those of other orbital growths; proptosis and the results of pressure upon the optic nerve, viz., swelling of the optic disc or atrophy. A diagnosis can sometimes be made by the removal of a little fluid with a hypodermic syringe, and the examination of it by a microscope, when the characteristic hooklets will be seen. The treatment which has been adopted for hydatids of the orbit is either excision of the whole growth, or tapping. In some cases in which the growth has been removed entirely, disastrous results have followed—the cornea has become ulcerated, and the eye suppurated; those who have had most experience of this class of case recommend repeated tapping, not total excision.

Before speaking of solid growths of the orbit, I would like to refer to the method of examining a

patient who is supposed to have an orbital tumour. The first symptom which will be noticed is proptosis, and the best way of determining the presence or amount of this is to seat the patient in a chair and stand behind him, directing him to look downwards; then by raising with the two hands the lids, and looking from behind and above, an accurate estimate can be obtained of the amount of protrusion of the eye. The next step is to notice the movements and position of the eye. In some cases, when the tumour starts at the apex of the orbit, the eye is displaced forwards, and not laterally or vertically. When the tumour grows from the walls of the orbit, the eyeball is generally displaced either laterally or upwards or downwards, as well as forwards. As an example of what the pressure of an orbital tumour will do to the eyeball, I show you a very large sarcoma which was removed from the orbit, and the eyeball from the same patient. You will notice how much the latter has become flattened by the pressure of the growth. Having examined for proptosis and displacement of the eyeball, the next thing is to feel for a swelling, by passing a finger between the lids and the walls of the orbit. If a swelling be detected, you must determine whether it is elastic, firmly resistant, or of bony hardness. Having found a swelling, you still cannot be sure that a growth is present. All these symptoms which I have mentioned—proptosis, displacement of the eye, and the presence of a firm mass—may be caused by a distended frontal sinus. The displacement of the eye in such cases is downwards and outwards, and the hard swelling is felt in the upper and inner portion of the orbit. It is often very difficult to distinguish between such cases and new growths in that part of the orbit. After satisfying oneself as to the presence of a tumour in the orbit, you come to the still more difficult matter of determining its nature. Tumours which occur in the upper and outer part of the orbit are most probably connected with the lachrymal gland, but not necessarily so. By the feel of the tumour one can often tell if it is cystic or of bony consistency, but generally a certain diagnosis cannot be made until an exploratory incision has been resorted to, and a portion of growth removed for microscopical examination. Even when this is done, if it is found that the tissue removed is

composed of round cells, it is very difficult to say whether the growth is a round-celled sarcoma or an inflammatory structure, such as a syphilitic gumma. A course of treatment with iodide of potassium is often of use in determining this point. A sarcoma of the orbit may start in the fibrous connective tissue, in the periosteum, or in the muscles. I show you one that started near the apex of the orbit, and spread up the sheaths of all the recti muscles. The whole of the eyeball, together with the tumour, was removed *en masse* in this case. Sarcomata starting in the periosteum are sometimes found to contain small pieces of bone, and are then called osteo-sarcomata. Ivory exostosis in the orbit may begin either in the walls of the orbit or in one of the sinuses around it, breaking into the orbit secondarily. The frontal sinuses is a frequent seat of an ivory exostosis, the tumour grows, extending downwards into the orbit, and upwards into the cranial cavity; the first thing noticed is the tumour in the orbit. If you operate on such a case, it is impossible to remove the whole growth, and operation on them sometimes leads to very disastrous results. Here is a specimen of an ivory exostosis of the orbit, which was removed in this hospital. The operation was an exceedingly tedious one, and occupied several hours, two or three Archimedian screws being broken in the attempt at removal. The operation was successful, so far as the removal of the orbital growth was concerned, but a few weeks afterwards the patient died of meningitis. At the post-mortem examination, the growth was found to extend into the cranial cavity as much as it had done into the orbit. In such cases operation is not advisable. There are other ivory exostoses which begin in the walls of the orbit, not in the sinuses around. These are sometimes very easily removed. They begin most frequently on the inner wall, and have a narrow pedicle, which can sometimes be broken through by the application of a moderate amount of force, and the whole growth removed. I show you a cast of an exostosis removed in this way.

It is very often necessary to remove the eyeball together with an orbital growth; when the object of the operation is to save the life of the patient a good seeing eye has sometimes to be sacrificed. The best way of excising the whole contents of the orbit is to make an incision

around the margin of the lids $\frac{1}{4}$ of an inch from the margin, then to dissect inwards between the skin and the tarsal cartilages upwards and downwards to divide the tarsal and palpebral ligaments, and then with a stout pair of curved scissors to cut back close to the wall of the orbit, dividing the recti muscles and the optic nerve at the apex; in this way the whole conjunctival sac, eyeball and muscles, can be removed *en masse*, as you see in this specimen.

I now come to the tumours of the eyeball itself, and will classify them according to the anatomical structures in which they start, taking first those of the cornea and conjunctiva. The cornea and conjunctiva are best taken together, because tumours most frequently start at the junction of those two structures, and it is difficult to say to which of them they primarily belong. The commonest form of tumours in connection with these structures are what known as dermoids. Dermoids are solid masses of skin occurring at the sclero-corneal margin, about the size of split peas. They are usually situated a little below the centre of the cornea, opposite the palpebral aperture, and are sometimes multiple; they are occasionally associated with that rare congenital abnormality known as coloboma of the upper lid. Patients who have these congenital dermoid growths usually come for treatment at the age of puberty, first, because it is then they begin to be interested in their personal appearance, and, secondly, because hair then begins to grow upon them, which causes irritation. The drawing of one which I show you here is copied from Wardrop's "Diseases of the Eye," and the author of that work mentions that when the patient's beard began to grow, hair commenced to sprout from the dermoid tumour.

These dermoid tumours are very easily removed, but a scar is always left behind, forming an opacity in the cornea. Occasionally, though rarely, cysts occur in the cornea—implantation cysts.

Malignant tumours of the cornea are fortunately very rare, both sarcomata and epitheliomata. They generally start at the sclero-corneal margin. Here I show you a specimen of a sarcoma starting in that position. Here is a sarcoma more of the conjunctiva than of the cornea, deeply pigmented, but not invading the interior of the globe. This

specimen is an epithelioma, presenting quite the characteristic appearances. If you have once seen an epithelioma of the cornea you are not likely to mistake it for anything else, the growth having a dead white appearance due to thickened epithelium on its surface.

Tumours of the iris are rare. The iris, however, is the most frequent seat of cystic growths of the eye. Cystic tumours of the iris may be divided into epithelial cysts, endothelial cysts, pigmented cysts, and cisticerci. I mentioned to you in my first lecture on injuries to the eye, how sometimes eyelashes are carried into wounds in the eye, and so, in the same way, sometimes pieces of epithelium from the surface of the lid, or from the surface of the cornea are carried into the eye and implanted into the iris, or into the anterior chamber. When so implanted, they act like grafts, and tend to grow and give rise to cysts, which are hence called implantation cysts. The cysts most frequently met with in the iris are found to be of such a nature they follow on wounds of the eye, and are lined with laminated epithelium. Some have a sebaceous, and some a clear fluid contents, I show you a cyst of such a nature in the iris. An eyelash was carried into the eye at the time of a wound, and though the lash was subsequently removed, its sheath remained behind; this grew into the cystic tumour you see in the iris. It is very important that these implantation cysts should be removed early and completely, because they tend to grow, and if any epithelial contents is left behind, it forms the nucleus of a fresh cyst.

Endothelial cysts of the iris do not necessarily follow wounds of the eye. They are lined by only a single layer of endothelial cells, and are probably due to the closure and subsequent distension of the crypt-like spaces which are found in the anterior part of the iris.

The third class of cysts of the iris are due to separation of the two layers of pigment-epithelium on its posterior surface. Sarcomata of the iris are very rare indeed. I have only seen one case, and that was in this hospital, when I was house-surgeon. One of the sisters in the ward asked me if I would look at her brother's eye, saying he had got a little black spot in it. On examination, I found a little raised pigment swelling in the lower and outer part of the iris, which I thought could be nothing else than a sarcoma. I showed it to

the other surgeons, who agreed as to its nature; and though the boy had perfect sight in the eye, we decided it would be better for him to have it removed, which ultimately was done. Microscopical examination confirmed our diagnosis, and showed that the new growth was beginning to invade the ciliary body, so that iridectomy would not have been of any use, as it would have been impossible to have removed the whole growth by such an operation. A case has, however, been recorded in which sarcoma was diagnosed, and the growth removed by iridectomy, no recurrence afterwards occurring.

On getting further back into the uveal tract, sarcoma becomes less rare. Sarcoma of the ciliary body is less rare than sarcoma of the iris, while sarcoma of the choroid is less rare than sarcoma of the ciliary body. The ciliary body contains three classes of structures—(1) ordinary connective tissue; (2) muscular tissue; (3) glandular tissue. It is therefore interesting to note that three classes of tumours arise from this part of the eye, viz., sarcoma, myoma, and carcinoma: all these tumours present much the same clinical symptoms, which are a certain amount of failure of sight and of pain. On looking into the eye you may notice, by oblique illumination, behind the lens and close to it, a raised mass, sometimes pigmented. There is generally some enlargement of the episcleral vessels in the neighbourhood of the growth, occasionally shallowness of the anterior chamber, and sometimes increased tension. The growth may extend forwards into the anterior chamber; if it does, it tends to separate the iris from the periphery of the cornea, and you then see a black line at the margin of the anterior chamber. That is a very distinctive sign of sarcoma of the ciliary body when it occurs; it looks sometimes very much as though there had been a traumatic iridodialysis.

Sarcomata of the ciliary body are sometimes accompanied by detachment of the retina, sometimes occur without detachment. I show you a specimen of a white sarcoma of the ciliary body, and of a melanotic one. The white growth did not appear to be of that colour clinically; a distinct black growth was seen behind the lens, because the pigment epithelial layer was stretched in front of it, and it was only when a section was made that it was found to be a white growth.

Coming now to sarcomata of the choroid. The symptoms first complained of by a patient with this form of growth is failure of sight, which is somewhat characteristic. The patient will often say that the failure began on one side, and then gradually increased, until ultimately vision was completely lost; ophthalmoscopically you may detect detachment of the retina, and, in some cases, behind the detachment a solid pigmented structure can be seen, rendering it unlike an ordinary detachment. In other cases no solid looking mass can be distinguished behind the detached retina, the reason being that besides the growth there is a large quantity of fluid in the sub-retinal space. In many of these cases the presence of the growth is not diagnosed until the symptoms of glaucoma come on. Many cases of detached retina are watched, and thought to be simple detachment, until pain and tension supervene. Then the eye is removed, and a sarcoma of the choroid is found. As a general rule, any eye with detachment of retina and increased tension should be excised, the probabilities being so great that it contains a malignant tumour. A sarcoma of the choroid may be white, or it may be pigmented. They frequently grow from a wide base which contracts into a narrow peduncle, and then expands into a rounded knob, the narrow pedicle marks the point where it has broken through the elastic lamina of the choroid, after which it fungated out into the rounded knob. In the same way, when a sarcoma gains exit from the eye and perforates the cornea or sclerotic, it begins to fungate out in a great mass, such as you see in these drawings. Sarcomata are very liable to start in the choroid of shrunken and blind eyes, and that constitutes an argument for the removal of all blind useless eyes; they being so liable to become the seats of malignant growths. If an eye with sarcoma of the choroid be removed fairly early, *i.e.* before an extra-ocular growth has formed, and before there is increased tension, the prognosis is fairly good. A large number of such cases get no recurrence, but you can never promise your patient that he is absolutely safe. I have known recurrence to occur ten years after removal of the eye for melanotic sarcoma. The secondary growths in the liver and elsewhere being also melanotic. Here I show you two specimens which illustrate a very extraordinary family history.

This specimen, showing a piebald sarcoma of the choroid was removed 22 years ago from a patient who at the time her eye first became bad was pregnant. The child was duly born, and a few months afterwards the patient died from secondary growths. The specimen was mounted, and put into the museum at that time. About three years ago, the daughter who was born after the removal of the mother's eye came here. She was then about 19 years of age, and was pregnant for the first time, and in the same eye as her mother she had a growth of almost precisely the same character, a piebald sarcoma, which I have here for you to compare. It shows the value of keeping specimens in the museum, and it is the sort of family history you do not often get, the after family history.

Nævi of the choroid have been seen, and nævoid sarcomata. This one which I show you is of a fawn colour, with large haemorrhages in front of it, between the growth and the lens.

We now come to growths of the retina. Cysts are sometimes met with in eyes which have been blind for a long time, and where the retina is detached, but they are usually only discovered on pathological examination, and are not of much clinical importance.

Glioma of the retina is a growth which occurs most commonly within the first three years of life, and is sometimes congenital. It never occurs at a later age than 13. This feature is in marked contrast to sarcoma of the choroid, which may occur at any age, and is most frequently met with after middle life. Glioma of the retina is an unpigmented growth, and the cells are always round, resembling the nuclear bodies in the granular layers of the retina. In them there is very little protoplasm around the nucleus, and they appear different from those seen in round-celled sarcomata. Gliomata of the retina are divided into two classes; sometimes they grow from the inner surface of the retina, and are then called glioma endophytum; sometimes they grow from the outer surface of the retina, which is detached in front of them—glioma exophytum. The symptoms differ in the two classes. In the first kind you look into the eye and see the ragged growth itself, sometimes with floating nodules in the vitreous; but in the glioma exophytum, you see immediately behind the lens, sometimes even with

the naked eye, the retina which has a smooth glistening surface, and appears white from the growth immediately behind it, whilst coursing over it the large retinal vessels are plainly visible. The symptom which the patient's parents most frequently first notice is a glistening appearance in the child's eye. Left to themselves, gliomata continue to grow, and expand the globe; sometimes they perforate it anteriorly, and give rise to large fungoid masses. In the older text-books this class of case was spoken of as fungus haematoïdes. Nowadays the eyes are excised before they reach that stage, and so that term has almost fallen out of use. If a glioma of the retina is excised sufficiently early, the patient sometimes gets no recurrence. At one time glioma was considered to be of so malignant a nature that every case which lived was thought to be an instance of mistaken diagnosis, and was spoken of as pseudo-glioma.

I have traced out the after history of a large number of cases of glioma, and know of several that have lived. I saw a patient nineteen years after the removal of such a growth, and he was then alive and well. Occasionally glioma affects both eyes; the second one may be implicated after an interval of several years; or both eyes may be affected simultaneously. I show you two eyes which were removed from the same patient, the second after an interval of three years for glioma of the retina. It is a ghastly thing to have to remove both eyes, but sometimes such a course is the means of preserving life. Investigation of cases of glioma shows that if a patient lives three years without recurrence of the growth, they never get a recurrence. That, again, is very different from cases of sarcoma of the choroid, in which, as I have said, recurrences may occur ten years after the removal of the primary growth.

The diagnosis of glioma is often exceedingly difficult. A large number of eyes have been removed for glioma which have turned out to have some other disease. There are three classes of disease which are liable to be mistaken for glioma of the retina.

The first is tubercle of the choroid. In these cases, removal of the eye does not matter very much; indeed, probably it is the best treatment for such a tuberculous eye.

In the second class, which it is more important

to distinguish, the appearance which simulates the gliomatous growth is due to a congenital abnormality. The central hyaloid artery persists, and remains patent, and where it breaks up into branches behind the lens, there is a dense fibrous membrane, which glistens and gives rise to a white reflex. The main point of distinction is that you can usually get the red reflex from the periphery of the lens all round when this condition is present ; whereas, in glioma of the retina the reflex will certainly not be seen on one side.

The third class of case, which is more frequently met with than the other two, and to which the term pseudo-glioma is more often applied, is one in which the eyes have been the subject of inflammation, a cyclitis, a retinitis, and sometimes a choroiditis, in which there has been a large amount of plastic exudation into the vitreous that becomes organized into fibrous tissue, forms a fibrous membrane behind the lens, and gives rise to a glistening reflex clinically. The main point of distinction from glioma in these cases is that when the fibrous tissue which is formed behind the lens has become adherent to the root of the iris, it draws it back at the periphery, and the anterior chamber becomes deepened in that situation.

Tumours of the optic nerve are very rare. I show you a specimen which was removed here. The symptoms which it gave rise to were as follows : proptosis, the eye seeming to be projected straight outwards ; there was no displacement of the eye upwards or downwards, or laterally. There was no restriction of its movements ; the ocular muscles were not at all affected, but there was well-marked atrophy of the optic nerve, and complete loss of sight. Tumours of the optic nerve differ very much microscopically ; some are myxomatous, some gliomatous, some sarcomatous, some myxo-sarcomatous ; in fact, nearly every surgeon who has recorded a tumour of the optic nerve seems to have found a different name by which to describe its microscopical appearances.

Castration for Hypertrophied Prostate.—

Kummel has performed castration for eight cases of prostatic hypertrophy, and also collected fifty-nine other cases. As a result of his experience, he holds that the operation is the true remedy.

(*Berliner Klinik.*)

WITH MR. ARBUTHNOT LANE IN THE OUT-PATIENT DEPT., GUY'S HOSPITAL.

Gonorrhœal Arthritis.

THIS young married man presents a hand and wrist considerably swollen, the swelling dimpling on pressure. Pain is greatest over the wrist joint, and flexion of this articulation causes severe pain. There is apparently some slight roughness on movement, as if the cartilage were inflamed. The patient tells us that he had gonorrhœa six weeks ago, which apparently spread itself over two or three weeks, and that this swelling then commenced very actively, the pain commencing between the thumb and first finger. There is no tubercular history in his family. His pulse and temperature are both normal. The causes of the condition which suggest themselves to me are gonorrhœa, rheumatoid arthritis, or tubercle. The history he gives, and the fact that he has had gonorrhœa so recently, make it practically certain that he is suffering from gonorrhœal arthritis, especially as his temperature and pulse are normal. In my experience it is unusual for the wrist to be affected in the course of a gonorrhœa as compared to other joints.

We had a very bad case of gonorrhœal arthritis in the ward, affecting chiefly the knees and elbows, which had persisted for eighteen months, and prevented the patient from standing or walking. We incised the knee and elbow joints, and cut out the abundant and bulky fringes of synovial membrane which were present, a quantity of fluid escaping at the same time, and placed a quantity of sterilized iodoform in each joint. This treatment was followed by a very marked improvement in the condition of these joints, so much so that we were able to get the patient out of bed. It is obvious that the wrist joint does not tend itself so readily to this form of treatment as do the hip and knee.

The patient before us will be admitted into the hospital.

Sarcoma.

This man, æt. about 50, has a large mushroom-shaped tumour attached to the alveolus of the lower jaw, and growing from the periosteum lining

the cavity about this carious tooth, which has been loose for some time. He has no enlarged glands. You will notice that the growth is pedunculated, and that there is no such ulceration of its surface as would suggest as a cause epithelioma; besides, they do not fungate as this one does. It is a sarcoma, and would be included clinically under the obsolete term "epulis." The treatment will be removal of the fang, and of the periosteum and bone from which it is growing, and the man will be taken in for that purpose. As an illustration of the remarkable fungating character of some sarcomas, I would remind you of a man on whom we operated some fortnight ago, who had a melanotic sarcoma as large as an orange attached by a pedicle as thick as one's little finger to the skin in the posterior triangle. This had been growing for four years, and under the microscope exhibited a typical melanotic structure. Yet the only secondary seat of infection was a lymphatic gland immediately beneath its point of attachment. The duration of melanotic sarcomas is extremely variable.

Cleft Palate.

This boy, æt. about 9, has a very complete cleft of his hard and soft palate; and I am asked whether a further operation now will benefit him. He was operated on for hare-lip when a few weeks old. As you are aware, the usual method of treatment in these cases is to do the lip when the child is a few weeks old, and then operate on the palate at the age of 3 or 4. Recently surgeons have recognized the importance of operating on the palate at an earlier age. That, I take it, is bad surgery. As I have pointed out very clearly on several occasions, the scientific plan in cases of cleft palate and hare-lip is to wait until the child is about 9 to 12 months old, the age varying with the nutrition and development of the child, when, thanks to the cleft in the lip and the gaping it allows of, you have a mouth large enough to admit of the operation on the palate being neatly and effectually performed. Do the palate first, and the lip afterwards. In my experience in these infants it is not always wise to operate on too much of the palate at once, but to be satisfied with bringing a portion together preferably in two places, and very much improvement results rapidly in the remainder of the cleft, facilitating further operative treatment. The lip can be attended to a month or so after the palate.

To operate on the lip at an early period, and to wait until the child is 2 or 3 years of age is to operate with a mouth often rendered very abnormally small and inelastic, so forming a small and unsatisfactory orifice to work in, and when the voice has been spoilt the nasal cavities and naso-pharynx are in an undeveloped condition, is unsatisfactory and unscientific. The operation, under these circumstances, does not give anything like the same comfort and advantage to the patient, and satisfaction to the surgeon. I believe the origin of the usual treatment was, perhaps, in this way. At the Hospital for Sick Children, I tell the parent to bring the child suffering from hare-lip and cleft palate back when it is 10 months or a year old, that the palate will then be operated on, and afterwards the lip, with an excellent result. They go away, and are told by Mrs. So-and-so to go to Mr. So-and-so, who operated on her child's lip at 3 weeks or so. I think the same occurs in private practice. In these cases, the mother feels some satisfaction in having had *something* done. I see that some surgeons are now adopting the course I suggested.

Necrosis of Femur.

This youth had typhoid fever, and during convalescence he got some infection of the lower end of the shaft of the femur, with subsequent necrosis. There remains a sequestrum, and a thick sheath of periosteal callus, discharging by two sinuses in the usual way. Why the posterior surface of the lower third of the femur should be so liable to infection does not seem very clear. It is possible that over-extension of the knee-joint may result in some haemorrhage beneath the periosteum about the attachment of the posterior ligament, and in a feeble patient in the supine posture such over-extension is very possible. The patient has relapsing swellings when there is an accumulation of matter. He must have the piece of bone removed.

Mastoid Disease.

Our next case is a little girl, who is brought on account of pain and slight swelling around the meatus, and the glands just below are also inflamed. The meatus is stenosed, and the mastoid is tender, but no discharge has been noticed except when the small abscesses in the meatus burst. She has a very high palate, with adenoid and some nasal obstruction. She has follicular abscess, probably with some middle ear trouble. Exploration can-

be performed except under an anaesthetic, and she will be sent into the hospital for this to be done, and for treatment.

Rickets.

You see the deplorable condition of this child—the distended abdomen with the conical protuberance at the umbilicus, and the curved limbs. The unsuitable name of rickets is bestowed upon these cases regarded from a skeletal aspect. It is not generally recognized that the changes comprised under the term rickets are not the only ones which result from the two frequent and unsuitable feeding in infancy. I pointed out that earlier results were those due to mechanical distension of the abdomen with gas, producing the typical broad half-paralysed yielding abdomen, with a gap between the recti and the protrusion so familiar to all of us; this increased intra-abdominal tension causing a yielding of the umbilical cicatrix, and of the obliterated processus vaginalis, with the formation of hernia and hydrocele. In a paper read before the British Medical Association, I pointed out that in the peritoneal cavities of these badly-fed children there is always a quantity of fluid, sometimes a considerable amount. In other words, associated with and in consequence of the disturbance of the intestinal function, there is a chronic peritonitis with effusion. This fluid adds to the increased intra-abdominal tension, and is itself displaced to form the hydroceles of infancy. Therefore, I need hardly tell you that to treat hydroceles and hernias in infancy by local means, as is usually done without attending scrupulously to the child's diet, is as utterly unscientific and ineffectual as it is general. I cannot impress this too strongly upon you. I regret to say, whether it is true or not, that in a considerable proportion of cases, the mother states that the frequent dieting is carried out at the advice of the medical attendant. Adenoids, bronchitis, imperfect dentition, constipation, diarrhoea, with the deformities that result from the resting postures, and many other conditions are all obviously chiefly produced by bad feeding.

Painful Swelling in the Sole.

This woman, æt. 30, complains of a painful swelling in the sole of her foot. I have fully explained to you, in my lectures on anatomy, that in the normal female foot the great toe is forced outwards against the others, through an angle of nearly 90° from the position occupied by the great

toe of the normal child's foot in the posture of activity; so that instead of that toe receiving and transmitting a very considerable proportion of the suprajacent weight, as it should do, the pressure falls on the heads of the second and third metacarpals, giving her pain as she walks, after having been on her legs a good deal, producing a corn beneath them. Please notice that the skin covering the under-surface of the great toe in this case is soft and silky, showing that mechanically it is of no service to the individual in its displaced position. Yet, as I said before, this condition of foot is perfectly normal to the fully-developed woman. It is quite obvious that it is no good trying to treat the local condition, but that you must look for, and remove, the primary cause. If the patient could afford them, I would order her a properly-made pair of boots with a toe post, the foot and toe being retained in a position of activity.

Varicose Ulcers.

You will notice this woman, æt. 60, presenting an ulcer on the lower third of the inner aspect of the leg. We have seen numbers like her to-day, and when you go up to the examination-table your examiner will worry you, as you have questioned me, as to what name should be applied to them. Now, Gentlemen, you will notice that in proportion as a surgeon knows nothing about the cause, and still less about the treatment of a condition, so he will classify it into many groups, giving to each variety a foolish and utterly meaningless name. Ulcers of the leg afford an excellent opportunity to surgery, from this point of view. Therefore, I will not ask you to give this ulcer a name, but I will endeavour to discover the several factors that determined its development, and try to gather from a knowledge of the causation some principle on which we can act in attempting to remove the effect.

As you know, the arrangements of the circulation of the leg are very ill-adapted for the mechanical relationships which we bear to our surroundings, or, in other words, to the state of civilization as it exists in larger towns, and, as I will show you, this is especially true of the woman. The blood which passes up through the deep veins of the leg is very much influenced by the action or inaction of the muscles about them. If the individual stands a great deal, retaining the muscles simply to balance the leg upon the foot,

and especially if the several toes and the several joints of the foot are cramped and their freedom of movement limited by boots, the action of the several muscles, which ought to drive in the blood, is in abeyance. This is still further exaggerated by forcing the great toe outwards in such a manner that the foot must assume the posture of rest, and by causing it to rub on a plane which is inclined to a variable, and often to a very considerable, extent by the heel of the boot.

This stagnating blood is thrown largely upon the superficial veins. Their arrangement is much worse than that of the deep veins, since under the influence of pressure the valves yield, and the saphenous veins become one long single column of blood, exerting a considerable and progressive pressure upon the walls. In savage life the erect position is not assumed for any long period of time, the individual sitting and lying down as much as possible.

This stagnation of blood is more marked in the case of the internal than of the external saphena vein; consequently, any condition resulting from it would naturally be more obvious in the area of distribution of the former. Stagnation of blood means impaired vitality, and consequently, of resisting capacity. The pressure exerted by the boot upon the veins of the foot serves to cause a more rapid charge of blood in them; but it is obvious that the skin on the inner side of the leg immediately above the boot is that in which organisms might most easily obtain a foothold, and that is the case. They produce to the naked eye an inflammation which the dermatologist calls eczema, and he adds to it the term "varicosum," to distinguish it from other varieties of superficial skin infection, and he will apply lotions, etc., to the part, and perhaps give the patient drugs. Later, the organisms obtaining a deeper hold on the part, a condition spoken of as an ulcer arises. As it varies in appearance, the terms applied to the ulcer are as variable as the forms of treatment, and are about as useless.

If the circulation in the limb is improved by placing the patient for a long period of time in the recumbent posture, the vitality of the part is sufficiently improved to be able to destroy the organisms which invaded it, and recovery takes place. On again resuming the erect posture, the same sequence of events arises as before.

Up till a comparatively recent time, the treatment of varicosity of the saphena veins, when sufficiently severe to require treatment, consisted in excision of portions of the dilated veins, or by multiple ligatures of the same parts. This was of very little use.

Professor Trendelenberg of Bonn suggested and practised ligature of the saphenous veins at their highest points, so releasing the superficial veins from pressure, and causing the blood to pass up through the deeper veins. I need hardly point out to you the vital importance of this measure, associated with other factors in the treatment of infection, resulting from diminished vitality of the part.

The old woman we have been examining does not desire to be operated on, and the circulation in the part will be improved as far as possible by pressure.

Hydrocephalus.

You scarcely need reminding that this infant of 8 months old is the subject of hydrocephalus. The treatment of these cases is generally unsatisfactory, and the prognosis is unfavourable. I have drawn off large quantities of fluid in some instances, but the head has generally filled up again. A morphia needle may be inserted through a sterile dressing, and in such cases, I have arranged to draw off a certain quantity during each hour by a hydraulic arrangement. Some did very well under this, but I never got any permanent benefit from the process. However, good results are recorded, and I presume those cases must be such as are on the border line between recoverable and progressive. One can easily imagine a case which would not get well except by tapping.

Gumma.

When this woman first presented herself, one side of her mouth and lips was invaded by a very extensive brawny mass, which had existed since July last, and produced a dreadful deformity and limiting her diet to slops. There were two or three spots of ulceration on its surface. I do not remember having seen anything like it. It was a gummatous condition, and it has subsided with remarkable rapidity under one week's treatment with mercury, arsenic, and a mixture of the iodides of potassium, sodium and ammonium.

It brings home to us once more the necessity of playing the trump card when in doubt, namely,

putting into the patient all the powerful germicides we possess, as a wrong diagnosis may mean a lot to a patient. It has cost this woman a great deal in more ways than one.

Antrectomy.

This young woman, 14 months ago, had antrectomy performed for chronic middle ear disease and facial paralysis, with the idea of curing her facial paralysis which had persisted for more than three years. Not only has her ear condition improved very considerably, but she can now twitch her face, and move it generally, which she was quite unable to do before the operation, though the paralysis has not been completely cured.

WITH DR. LEES

IN THE

WARDS OF ST. MARY'S HOSPITAL.

GENTLEMEN,—I had some cases of great interest to show you to-day, especially one of mediastinal tumour pressing on the trachea and oesophagus, and a case of total paralysis of the left third nerve in a young woman, with right hemiparesis and complete motor aphasia. But these must wait, for here is something which is urgent, which demands our immediate attention, and in my judgment calls for prompt surgical interference.

This boy, æt. 12, was admitted yesterday, and the history of his illness, as obtained by the clinical clerk, is as follows :

Eleven days ago he was seized with headache and vomiting ; he also commenced to shiver, and felt very cold. This attack was not very severe, and he was not confined to bed until three days later ; he even continued to run about for two days. The symptoms continued, however, and he suffered from diarrhoea for the next three days, and is said to have been "somewhat delirious." Slight shortness of breath became apparent. Four days before admission a doctor was called in and diagnosed a "bilious attack." The boy entirely lost his appetite, and his dyspnoea became much worse. On admission the dyspnoea was marked, and the boy complained of severe pain in the abdomen.

As we look at the patient as he lies, the dyspnoea is the first symptom to attract our attention. Observe how the alæ nasi dilate, and how the

upper thorax is raised at each inspiration. Noticing this, and remembering that the illness commenced with an attack of headache, sickness, shivering, and diarrhoea, with some delirium, we are inclined to suspect typhoid or influenza complicated with pneumonia, or possibly pericarditis. But on looking at the temperature chart, we find that his temperature yesterday was 100°, and that it fell this morning to 98·4°. That at once practically excludes pneumonia ; although you may meet with some bad cases of this disease in which the temperature is not over 102°, or even perhaps as low as 100°, you would hardly find pneumonia with a normal temperature. It does not, however, entirely exclude pericarditis, because in that disease the temperature is often much lower than in pneumonia ; still it makes even pericarditis less likely.

Now, if we turn down the bed-clothes and look at the boy, we see, in the first place, that he has a good deal of pain, and cries out when he is even lightly touched. The next thing we notice is, that although the upper part of his chest is raised with each inspiration, the epigastrium is almost motionless ; and the lower part of the thorax is comparatively quiet. We also see that what movement there is in his epigastrium is outwards during expiration, instead of during inspiration ; and if we very gently lay our hand over that region, we find that the movement is very slight, but that it is one of expansion during expiration. That at once shows us that the diaphragm is not acting, and here we have a most important fact with regard to the diagnosis. We next notice that his abdomen is somewhat swollen and is obviously tense, and that he seems to have pain in it. Just to eliminate the possibility of pneumonia or of pericarditis, we will rapidly but gently percuss his chest, and we find resonance everywhere except over the normal area of cardiac dulness. When we listen, we hear no cardiac rub or murmur, and inspiration can be heard down to the bases of the lungs in front and in the axillary regions ; and there are no rales and no crepitation or bronchial breathing. The boy is too ill for us to examine the backs of his lungs, but as far as we can gather from examination of the front, there is no affection of the lung or heart. We will now turn again to the abdomen, and ask the boy to point out the seat of pain. You see he passes his hand over

practically the whole abdomen. If I press gently, and equally, on the two hypochondriac and the two iliac regions, you see that the tenderness is decidedly greater in the right iliac fossa than elsewhere. The boy has been sick once since he came into the hospital, the vomit being liquid and dark green. The bowels have been opened once since admission.

These are the facts upon which we have to base our diagnosis. From the symptoms I have mentioned, I think it is quite clear that the boy is suffering from peritonitis. In some cases you may be in doubt between peritonitis and intestinal obstruction; sometimes the symptoms of the one may simulate those of the other very closely. It is not so here, because the bowels are not closed; but in many cases of peritonitis there is constipation, and peritonitis may produce symptoms almost exactly like those of intestinal obstruction. A good point of guidance is easily found in the action of the diaphragm. When the condition is due to intestinal obstruction, the diaphragm usually acts fairly well; when due to peritonitis the diaphragm is paralysed. Here the diaphragm is quite motionless, and that fact, with the pain, tenderness, vomiting, a low temperature, and a pulse of 132, prove, almost without doubt, that the boy has acute peritonitis. And you have probably already guessed, from the increased tenderness over the right iliac fossa, that the cause is very likely an affection of the vermiform appendix. At any rate, that is the most probable cause, though it is not the only possible one. The whole aspect of the case is too acute for a tubercular peritonitis, and there is no eruption to suggest that typhoid fever has caused perforation of the bowel, nor can we consider that there is perforation of a gastric ulcer, which at his time of life is exceedingly rare. Therefore the probability is very strong that his peritonitis is the result of appendix lesion. That being so, you will understand my remark on commencing, that the case was *urgent*, and that, in my judgment, it demanded *immediate* operative interference. Mr. Page has kindly seen the case with me, and agrees as to the necessity for operation. Permission has been obtained, and it will be carried out at once.

This is one of the cases of appendix inflammation (supposing my diagnosis to be right) which are *urgent*. Cases of appendicitis differ very much in intensity. Those which are extremely urgent

are usually due to the formation of a concretion in the appendix, and the perforation of the appendix by that concretion, resulting either in local abscess with limited peritonitis, or in a general peritonitis. With regard to that class of case, I should like to impress upon you, as strongly as I can, the necessity for surgical interference at the earliest possible moment. Every hour is of importance; they must never be left; they often run a short course, and are extremely dangerous. Delay of half a day may make all the difference between life and death. If you operate on such a patient within the first three days he usually has a fair chance of recovery; but if there is longer delay the chances rapidly diminish. When the patient is in the final stage of collapse, with pulse very rapid indeed, the chances of saving life are very small. Therefore cases do not bear waiting, and if you delay to make up your mind about the diagnosis, a fatal result is only too likely to follow.

There is another type of appendix case which is altogether different. It is the class of case in which there is some inflammatory condition of the appendix not due to concretion, but some catarrhal condition, sometimes recurrent, often causing the appendix to be thickened, or kinked, or altered in various ways, and attended with discomfort and pain, and vomiting perhaps, but not with the symptoms of acute general peritonitis. That class of case is of course on a totally different footing from the other class, and you will often find cases of this description recover even if very little is done for them; but they will recover more quickly if treated in a rational way, and I believe most quickly if you place an ice-bag over the local swelling. I showed you a case a fortnight ago—a woman in Carlisle Ward—who had very rapid relief from the local application of ice. I believe this treatment will reduce the duration of the illness by perhaps two or three weeks. Certainly ice gives them more comfort than any warm applications. Another difference between the mild and the severe types is, that in the milder cases one is often able to feel a local tumour. Gentle manipulation over the region of the appendix generally enables us to come down upon a tense, tender, localised swelling. But in the worst type of case you very often cannot feel anything except some tense resistance to the touch. The explanation is that in the very

worst type of cases, the appendix itself is not distended but perforated, and the abdominal muscles are so tight and painful, that even under chloroform little or nothing can be made out; yet the case may be in urgent need of operation. So that the absence of a local lump is not to be relied on in disproof of appendicitis.

Having drawn attention to those two types, I would add, with regard to the milder cases, that they are frequently recurrent; that is to say, the patient has an attack, from which he recovers; after an interval of good health, he has a second attack like the first: he may have a third, or a fourth, or even more. In some, the attacks are of diminishing intensity, and it is not worth while to interfere with them. But, unfortunately, they may grow more severe each time, and then it is desirable to consider whether it is not best to remove the appendix in the interval between two attacks, especially as a severe recurrence may occur when the patient is away from help.

I want to impress you with the distinction between cases which will not stand any hesitation, and in which immediate operation is a matter of life and death, and those in which the symptoms are comparatively slight.

Lastly, I might remind you of the case I showed you a fortnight ago, in which a man had a sausage-shaped swelling in the right iliac region with the long axis vertical, somewhat tender, yet without definite tenderness over the appendix itself. That was a case of typhlitis, as distinguished from appendix lesion or "perityphlitis," and it was due to an accumulation of faeces in the colon, which had set up inflammation in the neighbourhood, and there was considerable local pain about the cæcum. Ice relieved his pain, and the bowels were opened by large irrigation enemata, four pints in each, slowly inserted by an irrigator through a catheter while the hips were supported, half an hour being occupied each time. The lump in the ascending colon diminished day by day, until at last it disappeared and the man was well. Such cases usually occur in the second half of life. Cases of appendicitis, especially the most urgent, occur mainly in later childhood and early adult life.

[P.S.—The boy was at once removed to the operating theatre and anaesthetised. Mr. Page made a vertical incision over the appendix. As

soon as the peritoneum was opened there was a gush of liquid pus which rose in a jet higher than a foot above the level of the patient's body. From this evidence of tension, and from adhesions which seemed to bound the abscess, Mr. Page hoped that it was localised, and did not attempt to wash out the peritoneum. The boy was greatly relieved by the operation, but a few days later he developed parotitis, and it was clear that the abdominal wound was not draining satisfactorily. A second incision was, therefore, made posteriorly, and the abscess cavity thoroughly washed out. It was then possible to investigate the condition of the appendix, and it was found that half of this organ had sloughed. A ligature was placed round the base of the appendix, and the stump was removed.

Death occurred on the sixth day after the boy's admission, and at the post mortem examination general purulent peritonitis was found.]

Curettage for Puerperal Fever.—Dr. Rapin, of Lausanne, employs curettage for puerperal fever. The *débris* remaining in the uterus after labour and constituting the greater part of the lochial discharge aids in the development of septic endometritis. This is proved by the fact that in cases that have been curetted the lochia ceases in two or three days. Antiseptic intrauterine injections should be used when the temperature rises to 38° C., with foetid discharge. If a marked change for the better is not observed in twenty-four hours proceed to curette. Out of 34 cases Dr. Rapin had 6 deaths, several of which were in desperate condition when they came into his hands. In 13 cases cure was effected in three days; in 7 in less than eight days; and in eight, in twelve or fifteen days. In 1 case in which chronic puerperal fever without localization had continued thirty-two days a cure was effected in twenty-four hours. These facts show that in puerperal fever the process of infection is carried on in the uterus, and the symptoms are due to intoxication of the blood by toxalbuminoids and not to infection of the blood by microbes. The operation should be performed early. Any danger of rupture of the uterus, which is sometimes urged as an objection, may be avoided by proper precautions. This accident occasionally follows intrauterine injections.

(*Gazette des Hôpitaux*).

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 6, 1896.

CLINICAL NOTES.

WITH SIR DYCE DUCKWORTH IN THEWARDS OF ST. BARTHOLOMEW'S HOSPITAL.

Rheumatoid Arthritis.

THIS woman, æt. about 50, is the subject of chronic rheumatoid arthritis. The history is that the pains date from about two years ago, and that she has been unable to do her work since January of last year. She has had seven children in thirteen years. Several of her joints are very painful, including that of the lower jaw, but there is only slight effusion. She is in an acute phase of chronic rheumatoid arthritis, and these cases are often very difficult to diagnose. They very closely simulate gout and rheumatic fever; I have myself mistaken them for the latter, especially when several joints are inflamed. As to treatment, it should be borne in mind that chronic cases require chronic therapeutics; rapid improvement will not occur. Salicylates, as a rule, do not benefit this condition. The parts should be kept swathed in flannel, and large doses of iodide of potassium frequently give definite relief, especially when, as in this case, a number of joints are involved. This patient has been taking this, and has been having bedside baths. I shall now order a hot electric bath for ten minutes each day. A predisposing cause of the affection is frequent child-bearing, combined with hard work and insufficient food. These are factors in this woman's history. Progress towards health will be slow.

Post-Diphtheritic Paralysis.

This little fellow, æt. 4, is under our care recovering from post-diphtheritic paralysis. The strabismus is much less marked, but the knee-jerks are still absent. The heart-sounds are good and fairly regular. He swallows his food well, and it does not regurgitate. The knee-jerks in these

cases generally do return, but months and even years may elapse before they do so. I know a case in which this reflex remained absent for at least two years. It is usually felt that there is ground for anxiety while the knee-jerks are absent, but if they cannot be obtained for two years, during which there is no definite impairment of health, their loss cannot be a very serious matter.

The treatment is iron and strychnine, with good diet, in which a little wine is allowed. He is certainly markedly improving.

Pleuro-Pneumonia.

This young woman, æt. 23, came to the hospital on January 15th, this year, suffering from right pleuro-pneumonia of severe degree, with serous effusion. Her present temperature is normal; respiration 34; urine 1018, no albumen. She has not gained in weight. The pulse is soft; no murmurs are manifest. She has been tapped twice, and there is still an area over which the pleurae are adherent. She is very anaemic, but has made good progress. Her diet is a liberal one, and the treatment includes iodide of iron and nux vomica. You will notice that she has the "butterfly-wing" flush over the area usually occupied by rosacea. The moral indicated by the facts of this case is, that a pale, anaemic person is a bad subject for pleuro-pneumonia, or pleurisy; and, inasmuch as her pleurisy was on her right side, it raises the question to which I drew attention in my recent Lumleian Lectures, that pleurisy on the right side is more apt to be tubercular than on the left. At first I thought this patient was going to be tubercular, but I do not now think so. She will soon be able to leave the hospital.

Acute Rheumatism.

This case was admitted yesterday, and I at present know nothing of it, a position doubtless shared by others around me. Let us, therefore, see how far we can go towards a diagnosis without interrogating the patient. The first thing we notice is an air of listlessness and languor, with drooping of the eyelids, and the mouth partly

open. Probably you noticed that when we came alongside her bed she became flushed. Her tongue looks brown in the centre, the lips are dry, and the pupils large. You are aware that these signs point to a febrile condition. The respiration is tranquil, and causes the patient no difficulty. The position of the legs enables us to exclude peritonitis, the probabilities being in favour of general pyrexia. Now let us gain what information we can. The patient says she has had very severe pains in her legs, but that they have now left the legs and passed to the shoulders, and left arm and hand. All the pains were accompanied by swellings at the joints. At 7 years of age she had a somewhat similar attack, which kept her in bed for a month or six weeks, and rheumatic pains have troubled her at different times since then, but have not compelled her to lie by. One morning, about three weeks ago, she awoke with severe pains in her hands, which lasted three days, and then attacked her feet. On April 2nd, these pains suddenly became more acute and generalized, and a doctor attended her for a week before admission. A cough has troubled her since April 10th, and she has sweated a good deal. It may be noted that she frequently had a cough. We learn from the patient that she is æt. 23, and is already the mother of three children, the youngest of whom is æt. 5 months; her present illness came on while she was suckling the child. She has never had chorea. The family history is that her mother has had rheumatic fever six times, one brother three times, one sister five times, and another sister once. None of the members of her family have had chorea. The patient is fairly well nourished. On admission, her temperature was 104° , but it fell the same evening to 99° , after a dose of salicylate. The skin is hot and moist, the chest is well-formed, and its expansion is good. A lack of tone is perceptible about the heart sounds; there is a soft systolic murmur confined to the apex, probably endocardial. This may have been caused by the salicylate, though she has no tinnitus, and is not deaf. The percussion note is impaired over the right scapular region; there are some crepitations in front, on both sides, near the area of cardiac dulness, as well as early exocardial rubs on the left of the sternum. A soft localized murmur at the apex is almost always endocardial. She is having gr. xv of salicylate of soda every three hours. As

she has not had a motion for three days, I shall order her pil. cal. c. col. gr. v, which should be given every second or third night. A great many failures to relieve acute rheumatism are due to the omission of the calomel. The urine is acid in reaction, 1031, no albumen.

Alcoholic Peripheral Neuritis.

This woman, æt. 31, presents classical symptoms of peripheral alcoholic neuritis. She is unable to straighten her fingers, and efforts to carry one of her fingers to the tip of her nose result in erratic movements and complete failure to accomplish her purpose. Forced pronation causes considerable distress. You will notice that her legs are drawn up and everted, like those of a frog when lying on its back. There is the characteristic tenderness about the calves and the legs generally; moreover, these particular legs are very rickety. She has a bed-sore on the foot, which is getting better. A Macintyre splint is required for the legs. All her statements are untrustworthy, and, as usual with her sex, she denies any excessive drinking. Quinine and iron is about the best treatment, but these chronic cases occupy months in recovery.

Anorexia Nervosa.

This girl of 17 has been literally almost a skeleton, all her bones standing out in sharp definition. She has gained 1 lb. in weight during the last week. She goes out every day, and has twenty minutes' massage as often, but her temperature remains subnormal, and her bowels are constipated, the last motion being three days ago. The pulse is 100, and of improved tension. Urine 1018, clear, acid, and free from albumen. The tongue is clean, and she takes her food well. On her back she has several stripes of lineaæ atrophicæ well marked. I need hardly say that the patient should be kept in good spirits, and her food should be as appetizing as possible.

Polysarcia.

Here we have an example of an opposite condition to the last case, namely, obesity in a woman æt. 56. She came to the hospital three weeks ago, complaining of pains in her joints and back, but had been getting stout since her 20th year. She still complains that her ankles and knees are painful, the right knee "cracking" as she walks.

This is due to gout. Her urine stands at 1024, contains urates, and is scanty in quantity; reaction acid. She has lost 9 lbs. since admission, and can now stoop, but is not yet able to put on her own shoes. She is certainly making progress, her diet being regulated, and carbo-hydrate foods reduced.

Diabetes Mellitus.

The age of this woman is 37, and she is the subject of diabetes mellitus. She does not now complain of thirst, but has periodic visitations of excessive hunger. She cries out in her sleep; otherwise she rests fairly well. Formerly she had considerable itching, but that has now disappeared. 2 grs. of codeia were administered every twenty-four hours, this being subsequently increased to 3 grs. Itching was at one time said to be due to the impurity of the codeia, but itching sometimes occurs in diabetics who have not had the drug. She says she sweats a great deal, but that has not been verified. She complains, however, of severe attacks of flushing and burning heat in her limbs. She has gained 2½ lbs. in weight during the past week. There is pain in the abdomen, and general abdominal tenderness. The following are the figures of her case:—Urine passed per diem 32 ounces; it has been 60 to 70 ounces. Specific gravity of urine 1024; it has been 1035 and 1040, but as soon as she commenced 3 grs. of codeia the specific gravity dropped. Grains of sugar, per ounce 6·2; that also has decreased markedly. The urea has gone up. She has no ferric perchloride reaction, but there has always been a trace of albumen. We are justified in saying that the case is not now in the diabetic range. The tongue is not so characteristically red in this as in many cases, but there is great emaciation. The chest is clear, yet the breathing is a little shrill, which is possibly explained by the thinness of the patient. There is very little cough. Whenever, in these cases, there is progressive loss of weight, always suspect tuberculosis; it often supervenes when such patients are getting thin; a fat diabetic never becomes tuberculous as long as he is fat. As a rule, fat diabetics do not waste, or but very slowly; they are the chronic cases, while thin diabetics are the acute ones. This patient has got double soft cataract. The 3 grs. of codeia appear to act

satisfactorily, and the patient is progressing in the right direction.

Persistent Headache.

This single young woman, æt. 30, has been in the hospital a month. She has been the subject of headache for four years, which is said to have originated in an attack of influenza. That is the peg upon which nearly everything is hung now. The headache is chiefly vertical, and is increased at night and early morning; she also complained of pain and stiffness in the neck. She has had tinnitus, but that is now absent. The pulse is of high tension, her urine contains no albumen, is acid, and stands at 1014. Her refraction is good, but she is photophobic. There are no retinal lesions. Hearing is now good also, but a considerable time back she had pain and discharge in both ears. She has never been in a state of catalepsy, and is not fretting. Phenacetin did her no good. She sleeps naturally for four hours without a hypnotic. At first I thought it was a case of headache due to renal disease. She has lost 4 lbs. since last week. She has a seton in her neck. Setons are now made so aseptic that one does not get the same benefit from them as formerly; but I have seen marked improvement caused by them, especially in epilepsy. The question is whether it is not a hysterical headache, and whether the patient does not *enjoy* bad health. She has been having iodide of potassium and iodide of iron, but I shall now stop that and take out the seton, putting her on sulphate of magnesia three times a day.

Mitral Stenosis during Hemiplegia, leading to Cortical Embolism.

This patient, a woman, æt. 26, had hemiplegia on the left side a year ago; now she has it on her right, with aphasia. The right arm is quite limp; there is no rigidity. She is a little brighter, and more intelligent than she was, and eats and sleeps better. The temperature on the right side varies between 97 and 101°; on the left it ranges from 97 to 99·4°. The bowels are rather constipated; respirations vary from 36 to 48. On the right side the knee-jerks are exaggerated; on the left they are normal. The urine is cloudy, acid, and contains albumen, weight 1028. There is a presystolic murmur, followed by a systolic murmur. There are no musical murmurs, which

shows the absence of vegetations on the valves. The back is sound, and the sphincters intact. I notice a keloid growth on the sternum; the origin of these was a mystery until Mr. Hutchinson proved that they were due to irritation of the skin causing scars, probably induced by sinapsisms or blisters. The probability is that this patient has had both of the middle cerebral arteries embolised, the condition being really one of cerebral softening.

Chorea Traceable to School Pressure.

This girl, *aet. 12*, is the subject of chorea. She has had pericarditis, and her joints have been painful, but are not so now. She has almost lost the choreic movements, and now has pericardial and cerebral rheumatism. One can hear a double pericardial rub, and presystolic and diastolic murmurs. She sweats, too, but without any rise of temperature. At the apex there is a double mitral murmur and pericardial friction. The girl had been doing hard work preparatory to school examinations, and the sequence seems very clear to me—rheumatic diathesis, school-pressure, an attack of acute rheumatism, chorea, pericarditis. I am quite satisfied about the rheumatic nature of chorea; and those who are not, confess that the cardiac lesions in the two diseases are indistinguishable. This patient has pericarditis, and cerebro-cortical rheumatism or chorea. She is somewhat better, and little or no twitching is now observable. No motion has been passed for three days. She has been having biniodide of mercury ointment applied over the praecordia, and I shall now put her on quinine and iodide of potassium.

Arterial Pyæmia or Ulcerative Endocarditis.

This patient, a woman of *29*, was admitted on April 10th. She has been married sixteen months, and has had one child, six months ago, when she was in bed a month, and has not felt quite well since. Just before Christmas she complained of weakness and pains, which she called "rheumatics," and was treated for rheumatism. The pains, however, which were principally in the foot, knee, and shoulder of the left side, got worse, and she has been in bed since April 4th. A cough has troubled her six months, and she has come in suckling. There is a history of chills and sweats.

The patient does not know much of her early history, but says she has never been very strong. She had rheumatic fever six or seven years ago, which kept her in bed three months. Her father died at 45, of phthisis, and the mother succumbed to a "complication of diseases," which she is not able to specify. One brother has had rheumatic fever, and she has a brother and a sister alive and well. There are dark rings around the patient's eyes, and the mucous membranes are anaemic. There is no haemoptysis; she takes food fairly well, and the bowels are fairly regular. The pains in her left knee and ankle have passed away to-day. She has a scar resulting from a strumous abscess. The shape of the chest and its expansion are good, and the percussion note is good all over the front of the chest. The apex beat is not felt. A systolic murmur is heard at the apex and all over the cardiac area, and is transmitted to the back, at the angle of the scapula; this is much rougher now than on admission. There is also an accentuated pulmonary second sound. The spleen can be felt, and the splenic area is tender. The urine is smoky, weight 1020, acid, one-tenth albumen, and some blood. There has been no diarrhoea; the motion is semi-solid. Yesterday her temperature was 97° , rising to 100.4° in the evening; to-day it is 98° ; pulse 114, soft. Salicylates were commenced on admission. There is probably sclerosis of the mitral valve, and this view is favoured by the mitral regurgitation and the noisy pulmonary second sound. The patient's blood is evidently poor. Then there is the history of rheumatic pain in her joints ever since Christmas, and definite joint-pains on admission. The enlarged spleen and the presence of blood in the urine point to an infarct in the spleen and kidneys, emboli being cast off from the mitral valve. She may have chronic endocarditis, arterial pyæmia, or ulcerative endocarditis. Enterica also crosses one's mind. No one can yet speak with certainty here. The things to be on the alert for are embolic troubles. Salicylates may irritate her kidney. I shall order, at present, quinine and iodide of potassium three times a day.

Syphilis in a Girl *aet. 11*.

This unfortunate child is in a deplorable condition; she has the typical "peg" teeth, fibroid

nodules, and gummatous in the liver, enlarged spleen, signs of blockage of the portal vein, bony changes, enlargement of the ulna and tibia; she has also had keratitis and haematemesis. The treatment is large doses of iodide of potassium, and of the liquid extract of sarsaparilla, with oleate of mercury rubbed over the liver, and the administration of cod-liver oil.

A POST-GRADUATE LECTURE ON SLIGHT MULTIPLE NEURITIS.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, Feb. 26, 1896, by

W. R. GOWERS, M.D., F.R.C.P., F.R.S.,
Physician to the Hospital, and Consulting Physician to
University College Hospital.

GENTLEMEN.—We sometimes hear of “new diseases,” a term by which is meant diseases that are new to us. There is generally a tendency for novelty to have an undue influence, and the newly-recognised diseases are often seen where they do not exist. It is so, in some degree, with that which is pre-eminently the most important novelty among the diseases of the nervous system, peripheral neuritis. It is sometimes supposed to be the cause of symptoms that afford no justification for the inference. But, with all allowance for these cases, it is startling to note how frequent the disease is, how varied its causes, and that this must have been the case twenty years ago, when we knew it not. It is met with in all degrees, slight and severe—often so slight that the disease easily escapes detection. It is an example of this slight form that I desire to show you to-day. I should, perhaps, say that the case is an example of the relic of such an attack. It is not on this account less instructive, for the slighter cases of this disease cannot too often be brought under your notice, nor can you be too often impressed with the variety of its causation.

This patient, at the present moment, presents but one symptom. He has no knee-jerk; there is not the slightest responsive movement of the leg when the patellar tendon is struck. When you fail

to obtain the knee-jerk, you should test it again by the method of “reinforcement.” You probably know that it was first pointed out by Jendrassek, that if the patient energises the motor processes in the upper part of the spinal cord, as by hooking the fingers together, and pulling upon them firmly, and at the same time endeavours to divert his attention from his legs by shutting his eyes, or looking upwards, the knee-jerk is increased. It is well also to combine, with that, a little pressure of your fingers on the hamstring tendons. The great hindrance to obtaining the knee-jerk is the presence of a slight amount of voluntary muscular contraction. The pressure on the hamstring tendons seems to increase their relaxation, and also enables you to discern when it occurs. It is singular how many persons find it difficult to relax the muscles. Only yesterday I was testing a doctor, and the more he tried to relax the muscles, the more he failed to do so; there was always enough contraction to make it exceedingly difficult to obtain the knee-jerk. But in this patient, as you see, by no expedient can I obtain any result. When he stands up, and shuts his eyes with his feet together, there is perhaps a little more unsteadiness than normal, but the difference is not great. He now presents no other symptom. The motor power of the legs is good, and we cannot now find any defect of sensation.

If you met with this loss of the knee-jerk in an individual who had no other symptom, what would you think? The question is perhaps a little unreasonable, because a patient with no other symptom would not be likely to present himself to you. People do not usually go to a doctor for that which they are unable to discover for themselves, unless they are the victims of physiological curiosity on the part of some other person. The conclusion of many persons who find this loss and nothing else, is that, in the particular individual, there is naturally no knee-jerk. About sixteen years ago I related some cases in which I considered the knee-jerk was naturally absent. Since I published that paper I must have tested many thousands of persons, and I have never yet met with a single case in which the knee-jerk could not be obtained, and pathological conditions could not be traced; present or past. I have not, since then, met with a case in which the jerk seemed to me to be naturally absent. Therefore I look with very great

suspicion—indeed with more than suspicion,—on my earlier observation, and I am compelled to look thus also on the alleged instances recorded by others. I have seen many cases of the kind in which it could be obtained without difficulty, indeed I have not seldom felt distinctly uncomfortable when the characteristic movement instantly occurred in a patient brought to me with the assurance that it was absent. But I must also say this:—I have met with patients in whom I have failed entirely to obtain it on one occasion, but have succeeded on a second interview.

It is not easy to explain all these facts. One cause of difficulty, however, is the shortness of the patellar tendon in some persons, which makes it difficult to give a blow that shall so act on the elastic tendon as to cause the sudden traction on the muscle which is the actual excitant of the muscular contraction. I assume you know that it is not due to an influence on the nerves of the tendon: the tendon acts mechanically on the muscle, and the real question at issue is whether the reflex element is the actual contraction or the induced excitability of the fibres to the sudden mechanical stimulus. As regards the other sources of difficulty, it is important to remember that size of limb may make the crossed-leg position difficult, and involve a hindrance to movement; but this can be avoided by making the patient sit so that the legs hang vertically, care being taken not to mistake the swing for the jerk. Apart from this, the chief difficulty is the question of muscular relaxation, of hindrance due to contraction caused by mental attention, which most persons are absolutely unconscious of. I believe this contraction may occur just when the stroke is about to be given, so that the indications of perfect relaxation a moment before are fallacious. No expedient seems to prevent some persons being aware of the impending stroke; it may be only the sound of a movement, or some varied pressure elsewhere. At any rate, it is my conviction that the knee-jerk is always potentially present, save in cases in which there is some definite morbid change. When we are able to satisfy ourselves that it is absent, we can trace some cause for its absence.

It is so in this case. We have not to consider the possibility of natural absence, because there is a history of recent symptoms indicative of a morbid

state that affords a sufficient explanation of our inability to obtain the jerk.

He is a railway signalman, æt. 42. Four months ago he got a splinter into his little finger; this was followed by an abscess and swelling of the left hand, redness of the arm, and pain and swelling in the axilla. He was off duty seventeen days. As soon as he recommenced work, he noticed that his sight became dim after looking at an object for some time; then both hands became "numb," and tingled "like pins and needles." That was eleven weeks before his admission to the hospital about a fortnight ago. At the same time as the arm symptoms he noticed some "stiffness" in the calves, with tenderness. The word "stiffness" is often used to describe that which is not stiffness in the literal sense of the word, but a certain discomfort in the muscles which hinders their contraction, and the resulting movement. It thus has the same effect as actual rigidity, and so is described by the same word. Doubtless this was the condition in this case, since the calves are said to have been tender. A week after he gave up work his sight became normal, but then he noticed "pins and needles" in his legs and feet, as well as in the arms. As that sensation increased, his strength became less. He kept at his work, however, until a fortnight before admission, when he found, one day, that he could not rise in bed or stand. He had no shooting pain, and no affection of the bladder or rectum. This sudden disability was not persistent. During the fortnight before admission he rapidly improved, and became able to stand. There is nothing relevant in his previous history; there is no history of syphilis or of alcoholism obtained. On admission, in addition to the loss of the knee-jerk, which persists, he had an ataxic, staggering gait, liability to fall, and inability to turn quickly. He could not stand with his feet together and his eyes shut. Sensation is said to have been normal everywhere, with good localization, except in each little finger as far as the wrist. But a few days afterwards, on testing him very carefully, I found that there was distinct defect of tactile sensibility on the tips of all the fingers of the right hand. A very gentle touch, which could be felt on the left hand, was not felt on the right. A few days later the sensibility of the right hand had become perfectly normal. The ataxy also passed away, and, as you see, cannot now be recognised.

With the exception of the slight defect in tactile sensibility, the symptoms were bilateral, and that unilateral defect of sensibility was, I have no doubt, simply the residue of a defect which was before bilateral. With bilateral symmetry in symptoms you seldom have absolute equality in their degree on the two sides. On the side on which their severity is a little the greater, they last a little longer. Hence this fact does not invalidate my statement that the symptoms presented bilateral symmetry. Moreover, they were evidently symptoms corresponding to function. There was at one time brief weakness, but the most prominent symptoms were sensory—nerve irritation, tingling, numbness, and some sensory loss. The fact which this case illustrates, and which I would impress firmly upon you, is that whenever you meet with bilateral symptoms, greatest in the extremities of the limbs, corresponding to special functions of nerves, the first thing you should always think of is multiple peripheral neuritis.

Multiple neuritis, as the term has come to be used, is a special affection of the nerve structures. You may have a multiple neuritis which is quite a different thing. You may meet with unsymmetrical inflammation of several nerves, without any relation to function and without any true bilateral symmetry. That is always primarily an affection of the nerve sheaths, a perineuritis, an affection of the fibrous tissues, the nerve structures only being damaged when the inflammation passes to the substance of the nerve by the interstitial tissue, and affects the fibres in their course. The true multiple neuritis, to which the name is now given, is an affection of the nerve substance, an affection which is shown by degenerative changes in the nerve fibres themselves, in great excess of any evidence of interstitial inflammation or affection of the sheaths. It is this morbid process in the nerve structure, which has the special relation to function, and the special bilateral symmetry; it is this which has the marked tendency to preponderate at the extremities of the limbs. We can understand this fact, if we remember that the nerve fibres depend, for their vitality, on the nerve cells, of which they are the processes. The motor fibres are processes of the cells of the anterior cornua. The sensory fibres depend, for their vitality, on the nerve cells of the posterior ganglia. It is easy to

conceive that the further a fibre proceeds from the cell of which it is a process, the less will be the influence of the cell on its power of life and of vital resistance to that which would impair its nutrition. Hence it is that the fibres suffer at their extremities, and this is probably the chief reason why the fibres that have the longest course suffer most,—as the fibres of the limbs, and especially of the extremities of the limbs.

Remember that an influence which causes symmetrical effects of an acute character must be one that can reach all parts, and influence those most disposed, by their nature, to suffer—that is, it must be a blood state, or at least a toxic agent in the blood. Next, as a matter of experience, we find that agents which thus act on the nerve structures, and which we know, are always chemical substances, circulate in the blood. Metallic poisons often give rise to such symptoms, and when the agents are non-metallic substances, they are generally organic compounds, and many of these are products of low organisms. A conspicuous instance is alcohol, the most frequent cause of multiple neuritis, a product of the organisms of vinous fermentation. I have often reminded you of the significant fact that diphtheritic paralysis (which sometimes consists in a typical multiple neuritis affecting the extremities of the fibres, and even giving rise to loss of the knee-jerk and to ataxy), has been shown to be due to a chemical substance, generated from the albuminoes under the influence of a ferment produced by the diphtheritic organisms in the throat. Other facts justify us in assuming a like mechanism when multiple peripheral neuritis is related to a disease which we know, or have reason to believe, to be due to living organisms, for we find that the neuritis always *follows* the disease,—is a sequel of it. We meet with it thus after various acute specific diseases.

But we have also evidence of multiple peripheral neuritis succeeding various injuries. Does this agree with the other facts of causation? The instances that have been recorded have been cases in which the wounds did not run a healthy course. There was generally some evidence of spreading lymphatic inflammation; and we know that this form of inflammation is due to the introduction of septic organisms, and their development in the limb. Such low forms of inflammation do not occur in the

traumatic lesions that the surgeon produces, provided he takes strict antiseptic precautions. They occur in wounds into which we cannot doubt that organisms were introduced, and set up the special form of inflammation. The peripheral neuritis succeeds the primary process, as does that which we know to be the result of a chemical poison generated by the organisms of specific disease. I think we cannot doubt that such a process has been at work in this man, and that his transient but very definite symptoms were due to a toxic agent, resulting from the organisms which led to the inflammation. The facts are thus, you see, quite consistent with those of other forms and other causes. Remember that these forms of post-septic multiple neuritis vary much in severity. They have been, in some cases, so intense as to paralyse within a week the nerves of the organs on which life depends. They are more often slight, and are probably far more frequent than is conceived. It is not until we know what to look for that things are seen. As I said at the beginning, the nature of this man's symptoms would not have been suspected a few years ago. But now the significance of their character, and the fact that they may result from such a cause, are known, it is probable that similar symptoms will not infrequently be recognised. And remember that we are ever learning more and more of the wide range of causation of multiple neuritis.

All sorts of toxic influences appear capable of generating it, not only those due to agents from without, but also substances produced within the body in certain diathetic states, so that the practitioner should start with no narrow conception of its causation, he should start prepared for the unknown, he should be prepared to meet with the disease as the result of almost any toxic influence to which the human frame can be exposed. He should always bear in mind the absolute significance of the bilateral symmetry, and the specific disturbance of function of the nerves. He should remember also the significance of the loss of the knee-jerk that is due to the action of the toxin on the extremities of the afferent muscle nerves. For some reason those nerves seem especially liable to suffer, much more liable than the sensory nerves of the skin, and a very slight degree of impairment is enough to diminish the

reflex action on which the knee-jerk depends to a degree sufficient to prevent it.

Such a case presents no room for special therapeutics. The amount of the toxic agent was apparently small, and its effects were transient. Often, unfortunately, the quantity is such as to give rise to very severe changes, and the means of neutralising the influence, which must vary with the different poisons, belong to the future development of the marvellous therapeutic power that experimental pathology undoubtedly will provide.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM.

BY

JONATHAN HUTCHINSON, F.R.S., LL.D.

Reported by J. T. CONNER, M.D.

Extensive Morphœa Herpetiformis in a Pigmented Stage (Morphœa Nigra).

THE patient was a young woman, æt. 24, sent by Dr. Wickham of Canterbury. In either flank was a large, irregular, dark brown patch, extending upwards over the lower ribs, and downwards over the hips. The colour was exactly that of a pigmentary mole, and, as the texture of the skin did not appear at first sight to be altered, they might have been taken for huge moles. But, on closer examination, a band at the edge was found, distinctly hardened. It was of a lighter colour than the rest—yellow, and becoming white internally. The patches were almost perfectly symmetrical, and sloped downwards and forwards, parallel to the lower ribs, just like those of herpes zoster. There was a broad streak of similar appearance, running vertically over the lower half of the spine, and a few small patches, also arranged vertically, on the middle of the abdomen. The patient stated that the disease commenced five years ago as a small patch on the right hip, and the others soon followed.

Mr. Hutchinson insisted that, in this form of morphœa, which he designated herpetiformis, the condition could only be produced by causes acting

through the nervous system. A blood change could not produce lesions limited to particular zones with intermediate parts unaffected. The distribution resembled that of herpes, which owned a like cause. In some cases the resemblance was complete, where the morphœa patches were corymbiform, and limited to the area of a single nerve. Here many nerves were symmetrically involved, and the rounded outline, with absence of corymbiform arrangement, was, no doubt, due to slight spreading at the edges, and coalescence of patches. But it should be remembered that morphœa, unlike inflammatory skin diseases, remained localized, almost completely, to the parts first attacked. In this, again, it resembles herpes.

Pigmented morphœa had been designated "nigricans," but he thought this a mistake, as it was only a stage of the disease and not a distinct form.

Spreading Suppurative Folliculitis.

A man, æt. 55, brought by Mr. T. J. Hitchens, showed an oval purplish patch, about two inches in diameter, and raised half an inch, on the front of the right forearm. It was boggy and fluctuating. The skin was smooth, and perforated by a number of pin head sized orifices, from which issued, on pressure, clear serous fluid or sanguous pus. The hairs were all absent—had all fallen out. It commenced a month ago as a patch the size of a penny, studded with pimples, which became pustules, whilst the patch spread at the edge at the same time. Mr. Hitchens had carefully searched for the ringworm parasite, but with negative result.

Mr. Hutchinson said the disease obviously commenced as a folliculitis, which, according to the law of most inflammations, was infectious to similar structures to those first attacked. It was no doubt an analogue of the suppurative inflammation (kerion) which sometimes complicated tinea tonsurans.

Extensive Growth of Keloid in the Scars of Burns.

A man, æt. 30, sent by Mr. Openshaw, was severely burned, in the face and hands, by a gas explosion, eight months ago. The skin on the backs of the hands and fingers was entirely converted into scar. This was dense on the fingers,

and would permanently prevent flexion. On the hands were elevated ridges of pinkish keloid, running both transversely and longitudinally, so dividing the scar into a number of relatively depressed areas. It terminated abruptly at the wrist in a transverse roll of keloid, which was evidently advancing. This abrupt localization was explained by the nature of the cause, which was severe but instantaneous. The ears, and the skin of the face adjacent to them, were extensively scarred, and showed rolls and nodules of keloid everywhere.

Mr. Hutchinson explained that the condition of keloid differed entirely in nature and prognosis from the contracting scar of deep burns. The latter never formed keloid, because the corium, from which keloid always grew, had been completely destroyed.

Lupus Vulgaris of the Face with Rupialike Crust and neither Apple Jelly Growth or Nodules.

The patient was a woman, æt. 30, sent by Miss H. Webb, M.B. The disease began as a small sore on the upper lip fifteen months ago. From this it gradually spread over the middle of the face, including the nose. It was perfectly symmetrical. The margin was crescentic, extending from the angles of the mouth outwards on the cheeks and then inwards to the root of the nose. It was covered with a thick, yellowish brown, rupialike crust. Internal to this was a zone of scar, which was becoming white, whilst more internally, near the nose, was a brownish red deposit, which somewhat resembled "apple jelly." The nose was hyperæmic, extensively scarred and notched.

The diagnosis in this case lay between lupus vulgaris and syphilitic lupus. Nothing indicating syphilis could be found in the history. The patient was in good health, had been married twelve years, and was the mother of three healthy children. There had been no miscarriages. The rupial character of the crust, and the absence of nodules or "apple jelly," certainly were against common lupus and in favour of syphilitic. So was the fact that the disease began on the lip. But the fact that the disease had not travelled into the nares was against syphilis. Mr. Hutchinson thought the balance of evidence was on the

side of lupus vulgaris. But he could not positively exclude syphilis. He compared the case with that of a female with a similar thick pus-scabbed crust on the face, whose portrait he showed. In this case the disease was unquestionably non-specific.

Psoriasis becoming Universal.

The patient was a Russian Jew, æt. 46, brought by Dr. Dixon. The eruption began on the head three years ago, and thence spread to other parts. Dr. Dixon first saw the case two years ago, when it presented all the features of typical psoriasis. The patches had gradually coalesced, so that now only a few islets of normal skin could be found. These were near the axillæ and at the flexures of the elbows and knees. Here, at the margins, were a few small nummular patches which were still discrete. Everywhere else the skin was uniformly involved without the slightest indications of patches. It was much thickened, red and scaly. But the scales were duller and without the shining silvery characters. And there were no local heapings up such as occur with patches. In fact, the general appearance of the eruption was in no way characteristic of psoriasis, resembling rather pityriasis rubra or exfoliative dermatitis. But the presence of these small areas of perfectly normal skin excluded these diseases. In the latter there would be diffuse congestion of the whole skin, and not abrupt limitations. The face and palms of the hands were exempt. The head was very scaly, and its appearance resembled that of seborrhœic eczema. The patient was in good health.

General Erythematous eruption, approaching in character to lichen planus in parts.

THE patient was a man, æt. 63, sent by Mr. T. J. Hitchens. He was now in good health, but at the onset of the eruption, six months ago, he had suffered from "nervous debility." It was general but almost entirely a "limb eruption," consisting of deep red blotches, which were largest and deepest in colour on the legs. Here they were about the size of half-crowns, and almost purpuric in appearance. But they disappeared on pressure, leaving a slight fawn-coloured stain; and the eruption was not worse below the knees, as in purpura. The

patches on the arms were of lighter colour, and much smaller than on the legs. The eruption avoided the flexures of the limbs, and was most extensive on the extensor aspect. It was very pruriginous. The abdomen was almost exempt, but showed a few scattered, very small, somewhat papular spots. In the scapular region there were some flat, very slightly elevated, shiny papules, somewhat resembling those of lichen planus. Mr. Hitchens had observed similar spots on the fronts of the wrists, but they had now faded. The eruption was allied to lichen planus, but it was not possible to definitely name it. No cause could be ascertained.

Onychia affecting the finger-nails, almost symmetrically, in an infant, without apparent cause, but in association with intertriginous eczema.

The patient was a female infant aged a year and eight months. Two months ago, the skin on the front of the neck and in the groins became red and inflamed, with serous discharge from the former place. Simultaneously onychia of the third and fourth finger-nails of the right hand occurred. The inflammation then spread to the thumb-nails, and to the second, third, and fourth fingers and thumb on the other hand. Thus the index fingers were exempt on both hands and the middle finger on the right. But left middle finger was the least affected, which suggested that the corresponding right might be affected later, and the completeness of the symmetry merely delayed. The affected nails were thickened and irregular, and showed yellowish patches. The adjacent skin was red and swollen, but otherwise normal. There were no indications of eczema. The toes were not affected. The groins were erythematous, with a few outlying papules. In the neck the inflammation had much receded, but some redness still lingered at the furrows of flexure. The child showed obvious signs of rickets—large fontanelle, enlarged epiphyses, and beaded ribs, but appeared to be in fair health. There was no history or indication of syphilis.

Mr. Hutchinson had never seen any condition of nails like this in so young a child. He suggested that the eczematous inflammation of the skin might have caused the disease of the nails by contagion in scratching. He mentioned that an acute pustular onychia, in conjunction with pustular folliculitis,

was sometimes seen in children, without any history of syphilis. Here there was inflammation of the whole matrix, but not suppurative. There was nothing indicative of tinea in the appearances, but the contrary.

The treatment recommended was sending the child to the sea-side, and cod-liver oil. Locally, black wash or mercurial ointment should be used, or painting with strong liquor carbonis detergens, and followed by the ointment. But he said that the disease was very difficult to cure.

Keratoses of the hands and feet with Hyperidrosis.

A farmer, æt. 22, had suffered severely from this complaint almost ever since he began to walk. He stated that it began in the feet when he was three years old. Covering the heels were hard, bossy, yellowish plates of epidermis, which extended for about half an inch up the inside of the foot. A great mass covered the treads, and the whole of the under surface of the toes was similarly affected. The nails were much thickened, and corns also were growing under them.

In the hands the condition was much less in degree. The epidermis of the palms was diffusely thickened, and localised thickenings were present on the fingers. If a blister occurred after work, it always became a corn.

He was in good health, but much incapacitated by the disease, and troubled with profuse perspiration of hands and feet on exertion. When he had walked half a mile his feet became sore; but he could manage to walk five or six miles on soft ground, such as fields.

Mr. Hutchinson said that this was the most exaggerated form of keratoses he had ever seen. In cases such as this, where the corns were large, not only was there accumulation of epidermis, but the subjacent papillæ were hypertrophied. Thus the usual distinction between a wart and a corn—that the one consisted of hypertrophied papillæ and the other of accumulated epithelium—did not hold good. It should be stated thus:—In warts the hypertrophied papillæ grew through the epidermis; in corns they did not, but were concealed by the much greater accumulation of epithelium. But in smaller corns, such as produced by arsenic, there was merely epithelial accumulation, without hypertrophy of papillæ. Warts and corns did not occur

in the same subject. In this case there was a congenital peculiarity of skin, which produced an undue reaction to stimuli, in the form of callosities and profuse perspiration. The treatment recommended was change of occupation to a more sedentary one, and the wearing of thick soled boots, too large for the feet, and two pairs of thick socks.

Rodent Ulcer on the Nose.

A woman, æt. 53, brought by Dr. Fletcher, noticed a "lump" six years ago on the nose which became an ulcer two years later, slowly increasing ever since. On the left side of the bony nose was an elongated somewhat linear ulcer, about three quarters of an inch in length. It was much indurated, devoid of granulations, and adherent to the bone. The edge was undermined, hard, and bossy, and not "rolled." The adjacent skin was red and inflamed. There was no enlargement of glands. Mr. Hutchinson remarked that the rodent ulcer might occur on any part, but the most characteristic forms were seen on the face. He compared it with the crateriform ulcer, which occurred in the same parts. They were both epitheliomata. But the epithelial growth in the latter was considerable, attaining as much as half an inch on section, whilst the former was only about a tenth of an inch. Clinically they were in complete contrast. The crateriform was of rapid growth, infected glands, and did not recur on removal. The rodent of slow growth, never infected glands, and always recurred on removal, except in the very rarest exceptions, where operation had been done at a very early stage. Syphilis sometimes simulated rodent ulcer. A portrait of a tertiary ulcer on the forehead, with an elevated rolled edge like that characteristic of rodent, was shown; also one of chancre on the forehead simulating it too. The treatment recommended was free removal down to the bone. But it was not hopeful.

Leucorrhœa.—

½ Creolin.....	gtt. xxx.
Ex. hydrastis	fl. 3 iiss.
M. Sig. Two teaspoonfuls in a pint of warm water, to be used for one vaginal injection.	

—*Journal de Médecine, Paris.*

POLYP OF THE RIGHT FRONTAL SINUS.

Notes of a Case at the London Throat Hospital.

By GRIFFITH CHARLES WILKIN, M.R.C.S.

S. R., female, æt. 32, occupation, cook. The patient had suffered from discharge from the right nostril for five years. About eighteen months ago the discharge became thicker, and the breathing through the right nostril became difficult.

On the patient being examined on her first attendance at the hospital the right nostril was found full of large polypi. These were removed in the ordinary way. After their removal the discharge continued as before, of a distinctly purulent nature. On February 18th she was admitted to the hospital as an in-patient. Her condition was then as follows:—Her lungs, heart, and kidneys were healthy. She complained of a sense of weight and fulness on the right side of the head, right parietal region, no pain to the neighbourhood of the frontal sinus. She suffered from a distressing discharge from the right nostril, of a purulent, faintly foetid nature. This discharge was clearly traced to the infundibulum, through which a probe could be easily passed. The nostril was apparently quite free from polypi at this time. I determined to trephine the right frontal sinus, with a view to relieve this discharge.

As a preliminary step, to keep the nasal orifice of the infundibulum as free from diseased tissue as possible, I removed the anterior end of the right middle turbinate bone, and with it a large quantity of myxomatous tissue which had been quite invisible to the eye through the nostril. There was no rise in temperature after this operation; and after an interval of five days I trephined the right frontal sinus.

A small obtuse angular flap of skin was raised over the inner end of the right frontal sinus, the obtuse angle pointing outwards. A smaller flap of periosteum was raised, and a small trephine applied to the exposed bone. All haemorrhage was entirely arrested before the trephine was used. The sinus was opened a little outside the middle line. The whole sinus was found filled with polypic tissue, and the bone roughened. The whole of

this tissue was removed with sharp spoons, and the bone scraped. The infundibulum was quite patent, a silver probe being passed up from the nose into the sinus after the operation.

A small drainage tube was inserted into the sinus through the wound, which was then closed by three silk sutures, dusted with iodoform and dressed with blue gauze. After the operation the temperature rose to 100° F., and then became slightly subnormal. The sinus was carefully syringed each morning with a 1-1000 solution of perchloride of mercury, until it came freely into the nose and appeared quite clean. On the fourth day I removed two of the stitches, leaving only the one at the angle; the wound, excepting that part kept open by the drawing tube, healed by first intention. On the same day I shortened the drainage tube, leaving it out entirely on the eighth day. On the twelfth day after the operation a gland below the chin broke down and suppurated.

On the day after the trephining operation the woman expressed herself as entirely free from discharge in the nose, and at no time since has there been anything to speak of.

After removal of the anterior end of the middle turbinate bone there was no alteration in the discharge.

A CLINICAL LECTURE

ON

ACCIDENTAL HÆMORRHAGE.

Delivered at Aberdeen University

By R. G. MCKERRON, M.A., M.B., (Abdn.),

Assistant to the Professor of Midwifery, Aberdeen University; Obstetric Physician, Aberdeen General Dispensary.

GENTLEMEN,—The two cases which I am now to relate, furnish an excellent text for the subject to which I propose to direct your attention to-day,—accidental haemorrhage. Haemorrhage occurring during pregnancy is always an alarming, often a dangerous complication. In severe cases the risk to the mother is great, while the child is almost certainly lost. The condition demands careful supervision and, in its severer forms, prompt treat-

ment. By accidental hæmorrhage, it will be remembered, is meant the hæmorrhage that occurs during the later months of pregnancy from separation—usually traumatic—of the placenta occupying its normal site. The latter limitation is necessary to distinguish it from the so-called "unavoidable" hæmorrhage of placenta *prævia*, where the placenta, in whole or part, is implanted on the lower uterine segment.

The two cases referred to are of interest from the instructive differences they present, and are rendered all the more valuable for you from the fact, that in neither does the clinical picture exactly accord with the classical descriptions of the condition to be found in books. Text-book descriptions are analogous to composite photographs. They are the mean of all possible varieties, and bear often but slight resemblance to individual cases. They give the student the necessary standard, but a standard which in practice he will not find always observed. His view has to be corrected by the observation or by the record of concrete cases. In this lies the great value of clinical lectures.

Case 1.—Mrs. D.—, æt 23; in the beginning of the 9th month of 3rd pregnancy: on getting out of bed slipped and fell, striking left lower part of abdomen on the corner of a chair: faintness; and immediate discharge of blood from vagina, so profuse that in a few minutes clothing soaked, and on floor a pool of blood; over a quart must have been lost: when seen, less than an hour afterwards, midwife had put to bed: conscious, but pale and anxious: pulse soft, weak and rapid: tenderness over lower part of abdomen: uterine tumour of normal shape: no uterine contraction: external hæmorrhage stopped; no evidence of internal: os undilated: head presenting: placenta not to be felt. There was thus no doubt as to the condition. It is in the treatment of accidental hæmorrhage that difficulties are to be met, and that demands are made often on the promptitude, always on the judgment of the accoucheur. The treatment adopted was simple, the immediate risk, with the cessation of hæmorrhage, being over: an abdominal bandage was applied: rest and absolute quiet were enjoined: patient herself reassured: nurse left in charge with instructions to intimate at once if return of hæmorrhage, or any change in condition of patient. The onset of labour was pre-

dicted. No further bleeding however occurred, and the woman was delivered at term—three weeks later—of a well-developed living child. Placenta on examination found normal: no evidence of previous laceration.

Case 2.—Mrs. F.: eight and a half months pregnant: slipped and fell forcibly on knee, sustaining severe shock. For two days no symptom, save slight occasional abdominal pain, which was thought unworthy of notice. Suddenly, about forty-eight hours after accident, seized with severe flooding while sitting conversing with a friend. The bleeding lasted for a few minutes, and was followed by a slight ooze, at times increased: seen at 6 p.m., about an hour after: anxious and somewhat pale: pulse quick, soft, but of fair volume: slight discharge of blood from vagina: examination showed os soft and about size of a florin; cervix drawn up; head presenting; placenta not to be felt: normal abdominal tumour: uterine contractions at intervals of about ten minutes. Labour was thus begun. An abdominal binder was put on, and the progress of labour carefully watched: continuous ooze during the first stage, but never alarming: membranes ruptured early: labour artificially terminated by forceps at 2 p.m.: child dead: little blood lost after: slow convalescence.

In comparing these two cases, it will be seen that, in addition to following on a trauma, they have this symptom in common, that the placental separation was attended with discharge of blood from the vagina. In one, however, it followed immediately on the accident, while in the other there was no visible bleeding for forty-eight hours. Note, I say, no *visible* bleeding. Partial separation of the placenta was no doubt an immediate result of the accident, but, finding no egress, the extravasated blood accumulated between the placenta and the uterus. As it accumulated, it gravitated towards the os uteri, separating the intervening placenta and membranes. When the os was reached, forty-eight hours afterwards, the sudden flooding resulted. In this case, then, we had at first "concealed" or "internal" hæmorrhage, and the possibility of this following an accident in the later months of pregnancy must always be borne in mind. A further resemblance between our two cases may be noted. In neither was the hæmorrhage so severe as to place the patient's life in im-

mediate danger. In the worst cases, death may result before aid can be summoned, as happened here not very long ago. Between these fatal forms, and trivial haemorrhage, there are all degrees. In continuing our comparison, it will at once strike you that the case which at first was apparently the more alarming, proved the less serious. In the one the extent of the haemorrhage seemed to justify the prediction that labour would result, and that the child would be lost. In the other, the accident, though producing no symptom for forty-eight hours, brought on labour and was fatal to the child. The lesson to be learned is, that, no matter how slight the haemorrhage, the prognosis must be guarded, seeing that the nature and the amount of injury cannot be accurately determined. The external haemorrhage is no measure of the risk, indeed the most dreaded and serious form is where there exists no vaginal discharge to warn us of danger. In the favourable termination to both mother and child, notwithstanding the extent of the haemorrhage, lie the main interest and value of Case 1. The treatment adopted was mainly expectant. In Case 2, the partially dilated os left no doubt as to the onset of labour. As the bleeding was slight, and the constitutional symptoms neither marked nor increasing, no measures were taken to accelerate labour. The membranes were ruptured when the os was about one half dilated, in the hope, which was realised, of arresting the ooze. In severe and continuous bleeding the membranes should be ruptured at once. This, by increasing uterine contraction and retraction, and by effecting more direct pressure on the bleeding surface, is almost always beneficial. Should the haemorrhage continue and the patient begin to manifest the constitutional symptoms of great loss of blood, means should be taken to dilate the os, and to complete delivery as rapidly as possible. Dilatation may be performed by one or other of the hydrostatic dilators, but is best effected digitally. As a result of the haemorrhage the os is usually soft. Dilate first with one finger, then introduce two, sweeping them round and stretching the os till three can be admitted : when the whole hand can be introduced, turn either by the bipolar or by the internal method, and deliver.

Now a word as to internal haemorrhage. The quantity of blood effused is often enormous. It

may, you will remember, be combined with external haemorrhage. When not so combined the condition is usually much more serious, for the reason that the resulting shock is greater, and for the reason that the nature of the case is often doubtful. The two conditions with which it might be confounded are, rupture of the uterus and rupture of a tubal foetation. The diagnosis must rest on the duration of pregnancy ; the history of an accident ; increasing constitutional symptoms of haemorrhage ; abdominal distension, unequal in character ; uterine tenderness and irregular uterine contractions. A vaginal examination is absolutely necessary for diagnosis. Here the treatment must be immediate : a full dose of ergot, or better ergotin hypodermically : the membranes must be ruptured : abdominal friction should be tried, or a binder applied : and with these, if shock and collapse be not too great, artificial dilatation of the os, and rapid delivery. Stimulants may also be required. The after-treatment is that for shock and severe haemorrhage.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, North-West London Hospital,
April 15th, 1896.

Two Cases of Hemiplegia.

DR. HARRY CAMPBELL showed two cases of hemiplegia. The first occurred in a woman of 55. The seizure began with hysteroid symptoms ; the patient was somewhat violent, and attempted to bite those about her ; she then lapsed into coma, which lasted some hours, and left her paralysed down the left side. In three weeks' time it was almost impossible to detect any paralysis. Dr. Campbell diagnosed cerebral haemorrhage, resting this opinion chiefly upon the loss of consciousness and the almost complete recovery from paralysis, which is much less likely to occur when a vessel is blocked. He uttered a word of caution against always giving a grave prognosis in cases of cerebral haemorrhage, quoting the case of a lady, 86 years of age, who four years ago had a left-sided "stroke" which was attended with deep coma of three days' duration. At the present time it needed

a careful examination to detect any paralysis in this old lady ; she had still an acute intellect, and when last seen was engaged in composing a piece of poetry upon her grand-daughter's coming presentation at Court ! One must ever remember that the symptoms of cerebral haemorrhage may be largely due to the mere pressure of the clot, and that the symptoms thus arising may entirely disappear as the clot becomes absorbed.

The other case was that of a chronic alcoholic woman, æt. 48, with dilated left ventricle, frequent irregular thready pulse, anasarca, and enlarged liver. Her attack, also left-sided, was ushered in by premonitory symptoms ; there was no loss of consciousness, and there had been no recovery from the paralysis. Diagnosis : thrombosis. The distribution of the paralysis was interesting : the leg was slightly paralysed, the face considerably, the upper extremity *absolutely*. On asking her to make, with closed eyes, a vigorous effort to close the paralysed hand, it seemed to her as if it actually did close, though remaining quite motionless—thus showing that the muscular sense must, in part at least, be central in origin.

DR. LEONARD GUTHRIE agreed with Dr. Campbell that haemorrhage was probably the cause of the hemiplegia in one case, and arterial thrombosis in the other.

In the patient who had completely recovered, the haemorrhage was probably situated outside the internal capsule, and had produced the symptoms by pressure on the motor tract. As the blood clot contracted, the motor tract was relieved of pressure, and the symptoms subsided.

In the second case, thrombosis of some of the cortical vessels was suggested by the feeble, irregular, and dilated heart. It had probably produced necrotic softening of the arm centre in the right cortex.

The prognosis in acute cases of hemiplegia must always be guarded. A sudden cerebral lesion produced more widely spread symptoms than could be accounted for by the actual amount of structural damage caused.

For instance, complete hemiplegia might be produced by a lesion which only destroyed the motor tract of the arm. Here the face and leg might completely recover, whilst the arm remained paralysed. But it would be impossible to predict at first that this would be so. The amount of

damage done was always uncertain at first, and so also must be the prognosis.

Raynaud's Disease.

MR. JACKSON CLARKE showed a case of Raynaud's Disease. The patient was a woman æt. 45. She had suffered from the disease for nine years. The fingers of both hands were swollen and congested, and on the middle finger of the left side there was a sloughy ulcer as big as a shilling. The disease was looked on as due to functional nerve defect, which gave rise to vaso-motor spasm. It was brought to the notice of the surgeon as often as the physician. Gangrene of the nose, ears, or as in the present case of the fingers, or of the toes, leads the patient to seek surgical advice ; and in these cases it was important to recognise the nature of the affection, and to combine suitable internal with local treatment.

DR. HARRY CAMPBELL thought that paroxysmal haemoglobinuria was akin to Raynaud's disease, in that it might most reasonably be attributed to vaso-motor spasm.

DR. SIBLEY referred to the theory so elaborately worked out by Dr. Haig, that the real pathology of this disease was due to an excess of uric acid circulating in the blood, which under certain conditions produced a blocking of the capillaries, and so gangrene of the part.

DR. WM. BOULTING asked how it was that in paroxysmal haemoglobinuria venous stasis led to separation of the haemoglobin from red blood-corpuscles ? He also asked whether ichthyol had been tried in the present case ?

DR. MACEVOY also thought Raynaud's disease was due to vaso-motor disturbance, and in addition to what had already been said, adduced those instances of its association with hysteria where it often appeared after some strong emotion, its relation to blue œdema and erythromelalgia, which were occasionally associated with hysteria.

Some cases of hysterical Raynaud's disease, associated with polyuria had been recorded in France (Dr. Lévy and others), and their improvement or cure under hypnotism was in favour of the nervous origin of Raynaud's disease.

DR. LEONARD GUTHRIE adduced as other evidence in support of the vaso-motor or spasmodic theory of the disease, its association with spasmodic asthma. He related a case in which typical attacks of spasmodic asthma alternated with those of

"dead fingers." The two conditions never occurred simultaneously in this patient. Both were relieved by the use of vascular dilators.

In another case of true Raynaud's disease, gangrene of the tips of some of the fingers on both hands followed the application of extremely hot poultices. In subsequent incipient attacks gangrene did not occur, and relief was obtained by immersing the hands in luke-warm water, and by the administration of nitrite of amyl with nitro-glycerine and menthol. Extreme heat as well as extreme cold were known to produce constriction of arterioles, whilst moist warmth dilated them.

Excess of uric acid might possibly account for the symptoms of this disease, but a similar theory might also be conveniently applied to almost every disease under the sun.

In reply, Mr. CLARKE expressed agreement with Dr. Campbell as regards the pathological kinship of Raynaud's disease and paroxysmal haemoglobinuria. In the latter condition he thought the venous stasis, in addition to some unknown quantity in the blood, determined the solution of the haemoglobin. He doubted whether that unknown quantity might be uric acid. He regarded ichthylol as a most valuable drug, and had obtained good results with it in the treatment of lupus erythematosus, and so would expect it to be useful in Raynaud's disease, chilblains, and other kindred affections.

Psoriasis.

Dr. KNOWSLEY SILBEY showed a case of psoriasis in a girl, æt. 6. The lesions had been present for six months, and consisted of a few small roundish scaly spots, especially over the trunk; very few were present on the limbs, but a considerable number were to be seen in the hairy scalp. There appeared to be no other history of skin disease in the family. Dr. Sibley referred to some recent observations of Nielsen's, whose results with iodide of potassium were better than those with arsenic, especially so in the case of children, remarking on the fact that the lesions in children generally persisted for a longer time than in adults.

Osteo-Arthritis.

Mr. JACKSON CLARKE showed an advanced case of osteo-arthritis. The patient was an elderly woman, who first had a rheumatic attack thirty-three years ago. This first attack began in the right knee, and later affected the elbows and wrists. There appeared to have been some febrile dis-

turbance at this time. The second attack occurred three years later, and affected the arms. She was ill for several weeks, and then followed an interval of twenty years without rheumatic symptoms. The third attack showed itself in the knees and elbows, and the patient has not been well since. At the present time the elbows are the most troublesome joints. They were both marked by fluctuating swellings, which extended considerably beyond the usual limits of the joint. The joints were painful, and the bones could be felt to grate on one another. The great amount of distension could be accounted for only by supposing that besides the distension of the capsules of the joint there were hernial protrusions of the synovial membrane through the capsule.

Mr. Clarke showed several specimens illustrating the surgical bearings of the disease, which produced in some cases a dislocation of the joint, and in others virtual ankylosis from interlocking. In the present case nearly all the resources of medicine, regular or irregular, had been exhausted without any material benefit to the patient. As to the nature of the disease, the mode of origin in the present instance appeared to be of a truly rheumatic nature. There was no gout known to exist in the family. One of the patient's sons suffered from "chronic rheumatism."

Case of Abdominal Tumour.

Dr. SIBLEY also showed a case of abdominal tumour in an unmarried woman æt. 28. The patient had never had any serious illness. About two years ago she first noticed a small hard lump in the left groin, and this has gradually enlarged, and now occupies a considerable part of the abdominal cavity. She suffers no inconvenience except from the weight and fulness of the stomach. Menstruation always rather scanty, and coming on about every five weeks; has not changed one way or the other; she has never had any excess of haemorrhage. She appeared in good health, and had not lost flesh. The tumour, which was very hard on palpation, occupied the greater part of the abdomen; commencing in the left groin, it extended upwards almost to the costal arch, over towards right groin, and down into the pelvis. It was slightly moveable from side to side. From the history and physical characters of the growth, Dr. Sibley believed it to be a large solid tumour of the left ovary, probably a dermoid.

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A CLINICAL LECTURE ON AN UNUSUAL CASE OF HODGKIN'S DISEASE.

Delivered at Guy's Hospital, February 8th, 1896,

By FREDERICK TAYLOR, M.D.,

Physician to, and Lecturer on Medicine at, the Hospital.

GENTLEMEN.—This boy, æt. 8, has been under my care in Stephen Ward for some little time. You will see that he is emaciated, but that he has an enlarged abdomen, over which two or three distended veins are plainly visible, in which the blood appears to be coursing upwards. The liver is also enlarged, its dulness reaching to within an inch of the level of the umbilicus; and the spleen is slightly enlarged, projecting but little under the left costal cartilages. Besides this, the abdomen does not present any definite tumour, though there is a certain amount of fulness and resistance. In addition, certain of the lymphatic glands of his body are enlarged, viz., those in the neck, in the axillæ, and in the groin. Those in the neck are about the size of peas, those in the groin are somewhat bigger. The salivary glands are abnormal in size, and the parotid projects a little beyond the usual limit, besides which they are distinctly harder than normal. He has a moderate degree of anaemia—that is to say, he is a little pallid, but very much cannot be made of that point. During his stay in hospital he has had a certain degree of fever. I have the temperature charts for November and December, showing an elevation on some days to 101° and $101\frac{1}{2}^{\circ}$, while on one occasion it reached 103° . His temperature has also fallen as low as 98° , and the general character of this element has been decidedly irregular, with a mean above the normal. With regard to the condition of his blood—which is always a point of interest—it

presents no very great diminution in the number of its corpuscles; indeed, once or twice the corpuscles have been estimated to be above the normal— 105 per cent.; once or twice they have been 80 and 85 per cent.; two days ago they were 80 per cent. The white corpuscles are not in great excess—being 1 to 116 , which, though somewhat more than the physiological figure is not sufficient to constitute leukocytosis or leukocythaemia. The haemoglobin was estimated at 100 per cent. on the 6th inst., and, I think, previous examinations gave a corresponding figure. So that there appears to be no diminution of haemoglobin, there is no serious excess of leukocytes, and the blood is not very materially altered.

Moreover, with regard to anaemia, you will see that the colour of his lips does not suggest it, but it must be remembered that this is complicated by the obstruction in the circulation to which I have alluded, which may give the lips a persistent dark hue. I would also refer to the slight tendency to dyspnoea, which would correspond with the congestion, and is possibly due to some enlargement of the glands of the thorax coexisting with a like condition in the abdomen.

Now, in respect to diagnosis of this condition, we, of course, need to know something of the history of the illness. He was admitted on September 15th last, for pain in the left groin and swelling of the leg. Two months before admission he noticed a large lump in the left groin. It was not painful, and he was told to rest the limb, which he did not do; then, although the lump began to disappear, he got worse, and a change in the country gave him no benefit. When he came into the hospital ward much the same state was noticed as is present now, with very little alteration. On admission, he lay on his side with his legs drawn up, both thighs swollen, especially the left; the veins over the groin distended; enlarged glands of both groins, both axillæ, and in the cervical region, which were described as hard and "shotty," freely movable under the skin, and not adherent to each other. The glands in the groin

were tender ; there was no enlargement of the tonsils ; the superficial veins of the thorax and abdomen were distended, the course of the blood being upwards. On examination of the blood the red corpuscles were said to be 110 per cent. ; no excess of leukocytes. The lungs normal, except rather harsh breathing on the right side. The optic discs were normal. He was in Clinical ward two months, where he was put under certain treatment, and improved in ways I shall mention. Then he went out for a time, but returned to the hospital within a week. Between September and November, the size of the external glands undoubtedly diminished, especially those in the cervical and axillary regions. Shortly after his re-admission the enlargement of the salivary glands took place, and he had considerable enlargement of the parotid and submaxillary glands, so that they are felt to be hard and firm. I have photographs here showing the condition in December, which enable us to verify the fact that the enlargement is not now so pronounced, a change having been observed during the first week in January. Those changes may be considered as to his advantage ; but, conversely, we find a general deterioration of his other conditions—the abdomen is decidedly larger than it was, so is the liver : while the tinge of jaundice is a comparatively recent phenomenon. Moreover, the veins are distended very much more than formerly, and emaciation progresses. He is altogether weaker and less vigorous than he was.

The question arises, What is the nature of this case? Almost all the conditions I have alluded to point with tolerable certainty to Hodgkin's disease, or lymphadenoma ; and I do not think anyone who has seen him has expressed a contrary opinion. In stating what Hodgkin's disease is I want to be a little precise, because very generally there is some confusion in the minds of those who have not seen many of these cases between Hodgkin's disease and leukocythaemia. These two diseases do run on very similar lines, and cases occur in which it is difficult to say whether one or other is the predominant feature of the case ; but there are typical cases in which no mistake should be possible. When one meets with a disease which is associated with a name like this, it is interesting to go back and ascertain

t conditions the discoverer first really de-

scribed. The first cases which Hodgkin published were described by him in a paper entitled, "On some morbid appearances of the absorbent glands, and of the spleen." What is the appearance of the glands? They are enlarged ; they are in a condition of hyperplasia, or hypertrophy, or over-growth of tissue of the same nature as the normal gland tissue. This is a very delicate connective tissue forming a reticulum, the meshes of which are occupied by lymph cells, or lymphocytes. In some cases the reticulum is not excessive and is not thickened, while in other cases the reticulum is thickened and the leukocytes are in a much smaller proportion. In the first of these conditions the glands are comparatively soft, in the second, hard. Thus the hardness varies in degree, according to the relative proportions of connective or reticular tissue and of leukocytes. The glands are nearly always detached from one another, at any rate in the early stages, without tenderness or inflammation, or redness of the skin over them, and they move freely under the skin. Later in the disease it is true that, in some cases, there is matting together, or implication of the surrounding tissue, not by an inflammatory process, but by the perforation of the gland capsule and the consequent infiltration of these leukocytes into the connective tissue. That is to say, there may be an invasion by lymphatic growth, but not an inflammatory exudation. Any of the glands may be involved ; you may meet cases in which only the cervical glands are affected, or those of only one side of the neck, while in other cases every group of glands in the body may be implicated. External masses of glands are frequently involved, and sometimes the retro-peritoneal glands, or the mediastinal or bronchial glands. It is probable that such is the case here. Another point is, that as there is no adhesive inflammation of these glands, so also there is, as a rule, no suppuration of the glands in Hodgkin's disease. In a few cases caseation takes place, and suppuration may occur, but those must not be regarded as uncomplicated instances of Hodgkin's disease. To conform with modern ideas of caseation we must look upon cases in which this occurs as those in which the tubercle bacillus has invaded glands previously affected with Hodgkin's disease. I do not see any improbability in this.

Now we come to the spleen. The condition of

the spleen was also one of moderate enlargement, but coupled with this was the presence of a number of white bodies, which Hodgkin likened to tubercles. A typical spleen of Hodgkin's disease presents a number of round or oval white masses scattered throughout its substance, varying in size from $\frac{1}{2}$ to $\frac{1}{2}$ inch in diameter, and contrasting very strikingly with the dark red-brown colour of the normal spleen. You will note that enlargement of the organ is not referred to in the definition, and, commonly, the spleen is not very much enlarged in this disease. The white bodies are nothing more than masses of lymphatic structure, like the structure now called lymphadenoma. One of the names which have been given to it is "hardbake spleen," in conformity with the desire apparently possessed by the older pathologists to name diseased conditions after some article of food. I have given you a short statement of what Hodgkin's disease was, but, of course, the increased opportunities for observation afforded by fresh cases add to our knowledge. One of the symptoms is fever; the majority of cases of Hodgkin's disease are accompanied by some such irregular fever as I have indicated to you, not a septic fever, nor one characterised by oscillations of considerable degree, but a fever varying in different cases; and one that sometimes goes on for a certain time—three, five or six weeks; then remits entirely, and is subsequently renewed. Then the anaemia has, at different times, been made much of. Patients are commonly anaemic; in many of the most typical cases there is profound anaemia, and cases are recorded (the condition of the blood having been carefully noted) in which the corpuscles have been found to diminish to 50 and 60 per cent., and the haemoglobin in corresponding degree. So that anaemia is a characteristic feature, and has given rise to some of the names which have been employed, such as *anaemia lymphatica*. Let me impress upon you the fact that the anaemia of Hodgkin's disease is independent of the leukocytosis. There are, indeed, conditions which seem to form links between the two diseases, but those combined cases are by no means so frequent as the typical cases of either disease, and, as a general rule, we may say that typical cases of Hodgkin's disease are independent of real pronounced leukocythaemia, in which the leukocytes are in very great excess.

Now the enlargement of the glands leads us on to consider the infiltration of the surrounding tissue, or of the connective tissue in between the glands, which sometimes occurs by growth beyond the limit of the gland capsule. This is an important feature, especially in those cases where the growth is situated in the interior of the body, and where, by its continued enlargement, it may lead to very serious and disastrous consequences. In this patient we seem to be witnessing the effects of pressure upon the veins, the distension of the superficial abdominal veins being probably due to the pressure of enlarged abdominal glands upon the vena cava; and if a similar pressure occurs in the mediastinum you know that serious results may ensue, because of the number of important vessels and structures that lie there, such as the oesophagus, the trachea, the large veins, etc. There is also another way in which lymphadenoma appears beyond that involved in the statement of the disease which I have read from Hodgkin's original paper. These lymphadenomatous deposits may appear in other organs than the spleen, viz., in the liver and the kidneys. Further, it may occur in parts of the body independent of the solid organs, and independent of the gland structures. I remember one case particularly which I published in the "Pathological Transactions," illustrating the occurrence of masses of lymphadenomatous structure under the visceral pleura, or between the pleura and subjacent tissue. The lungs were compressed by effusion in the course of the pleurisy, and some adhesions had formed and were stretching across from the ribs to the lung; in the middle of these fibrinous bands were rounded nodular growths of lymphadenomatous structure. Other similar cases have been noticed and recorded.

As to the course of the disease, that depends on two or three factors. To some extent it depends upon the degree of enlargement of the glands, because there are mechanical difficulties arising from the pressure exerted by the growths, though the trouble from this cause is much less in enlargement of the external glands. Still, some striking results are sometimes seen from the enlargements of glands, which may even fill up the angle between the head and the shoulder, though even then the inconvenience to the patient

may not be very serious. It is scarcely necessary for me to say that if such a degree of enlargement occurs in the interior of the body, grave trouble is certain to ensue. Then, the anaemia may progress to such an extent as to constitute a formidable factor in the patient's condition, and this is undoubtedly one way in which the sufferer may be brought to an untimely end. Then there is pyrexia; the patients lose strength, perhaps dropsy supervenes, and later on inflammatory affections, such as pleurisy or pneumonia; and death occurs through exhaustion, or in consequence of those conditions which are secondary to inflammatory states.

The duration of the disease is variable, and may be of any length from six months to two years. This boy has been ill about seven months, and I think it is doubtful whether he will survive another seven months, but it is impossible to say positively.

I now propose to refer to the points in which this case differs from typical ones. Firstly, this differs from many in the degree of anaemia; that is a very noticeable point, because this boy's anaemia is very slight—a fact I have already shown you early in this lecture. I warned you as to the recognition of anaemia from the lips alone, because of the venous congestion, or the tendency to it.

The next point concerns the size of the lymphatic glands. The external glands—those alone about which we can speak with certainty—are not nearly so large as one sees in most cases. The relative degree of implication of the interior and exterior glands varies a good deal; sometimes both exterior and interior glands are involved; sometimes in very unequal degree. This case may present a much more serious implication of the deep glands than that observed in the exterior.

The third point relates to the condition of the salivary glands. I think that is a rare condition; I have not seen it mentioned in medical works, though I have heard of a similar case. I suppose the salivary glands are infiltrated with the same kind of lymphoid growth as that we have been discussing. Some years ago I showed one of those mixed cases, in which there was leukocythaemia associated with lymphoid structures in various parts of the body, and in that case there was general infiltration of the testes by a lymph-

adenomatous structure. It seems to me that the parotid and maxillary glands are most likely involved in the same way, notwithstanding that those glands are now smaller than they were a short time ago. We have examined his testes, and they appear to be normal.

With regard to the names of this disease, lymphadenoma is often used in England as an equivalent of Hodgkin's disease (Hodgkin was one of my predecessors in this hospital). The disease is also called malignant lymphoma. A name used by German writers is pseudo-leukæmia. I think that names which simply indicate what the disease *is not*, lack precision, and are apt to mislead. To say the disease is pseudo-leukæmia is simply to say it is not that which we know it is not, without helping us to determine what it *is*.

Now with regard to diagnosis, the most prominent features, as a rule, are enlargement of the glands and anaemia. Most frequently it is the first of these symptoms which induces the patient to seek medical advice. Therefore we have to consider what other diseases which present enlarged glands are liable to be confounded with Hodgkin's disease. First, we have to remember the possibility of swollen glands being tuberculous, or scrofulous, or strumous. These affections, unfortunately, are frequently encountered, and the development may be proceeding in different parts of the body. Moreover, Hodgkin's disease and tuberculosis occur at about the same period of life—early, or early-middle life—though they may occur in quite young people. Still, a slight distinction may be drawn in this respect, as Hodgkin's disease may be set down as usually occurring between puberty and 30 or 35, while tuberculosis occurs also earlier. As distinguished from Hodgkin's disease, tubercular glands are generally more isolated; they commence more as a local disease, but this distinction does not universally hold, because in Hodgkin's disease only one mass of external glands may be enlarged. Nevertheless, in a great number of cases of Hodgkin's disease, the glands all over the body are very soon involved, whereas, in strumous and tubercular glands, the mischief spreads more slowly, reaching the other glands only after a long interval. Again, in Hodgkin's disease, the change takes place without any inflammation, without caseation or suppuration or implication of the skin, and without any need for

surgical measures, unless these should be deemed expedient for the total eradication of the disease.

I need not refer to family history; in such cases, as a rule, this is not conclusive. Hodgkin's disease is comparatively rare, and there is no known reason why it should not occur in people who have a tubercular history. Therefore, a tubercular history may mislead you.

The next point is the differentiation of Hodgkin's disease from syphilitic glands. You are aware that enlargement of the glands occurs in secondary syphilis, commonly the posterior cervical glands, which is not the especial seat of tubercular enlargement, nor of that due to Hodgkin's disease. But there are cases of syphilis presenting a much more general glandular enlargement, including the axillæ and groin, and this increases the difficulties of diagnosis, especially as they are inclined to behave like lymphadenomata, that is, they may neither suppurate nor mat together. The history may help you to arrive at a diagnosis, and you may be helped to a correct opinion by treatment, because you know that syphilis is amenable to treatment by certain drugs, namely, mercury and iodide of potassium.

I must now say something more about the relation of lymphadenoma or Hodgkin's disease to leukocythaemia or leukæmia, because they have been frequently confounded. But my task of drawing a distinction between two diseases so closely related is rendered difficult by the scant definite knowledge we possess of either. Still, the typical forms of the diseases admit of a fairly distinct differentiation. Leukocythaemia means the presence of *excessive* leukocytes in the blood; a moderate increase would be sufficiently indicated by the term leukocytosis. Leukocythaemia is always associated with some disorder of one or more of the blood-forming organs, and subdivisions have been made on this basis, thus:—

Leukocythaemia splenica, cases in which the spleen is enlarged.

Leukocythaemia lymphatica, in which the lymphatic glands are enlarged.

Leukocythaemia myelogenica, in which there is alteration in the condition of the medulla of the bones.

It is now generally held that medullary leukocythaemia does not occur alone, but usually in

association with the splenic form; so that the cases are divided into the spleno-medullary and lymphatic varieties. The great distinction between Hodgkin's disease and leukocythaemia is that in typical cases of the former there is no leukocythaemia, while in the latter, of course, there is. I wish you to bear this point in mind, because diagnoses are sometimes made upon insufficient data, and I have known cases in which leukocythaemia has been diagnosed simply by reference to the spleen and the presence of anaemia, without the blood having been examined at all. That is not a safe thing to do, and the examination of the blood in all these conditions is a point of great importance. In leukocythaemia the spleen is very large indeed, without deposits, whereas such excessive enlargement is quite uncommon in typical uncomplicated Hodgkin's disease.

Again, it should be remembered that Hodgkin's disease might produce mediastinal tumour alone, by pressure, and such mass may be regarded as sarcoma or other malignant growth instead of lymphadenoma.

Finally, I will say a few words as to treatment. The treatment of lymphadenoma is unsatisfactory. In the majority of cases the patients die. Some do improve, and it is possible that some cases have even been cured when taken in hand early. Many drugs have been tried, one of which seems to have an effect for good in allied diseases, namely, arsenic. It has been successfully employed in some diseases of the blood, of the glands, and of the skin, such as lymphadenoma, leukocythaemia, pernicious anaemia, ague, pemphigus, psoriasis, and eczema. But to get the full benefit of arsenic it must be pushed—used in progressively increased doses up to twenty or thirty minims, or even more, three times daily, if such doses can be borne without inconvenience. This boy certainly experienced benefit from arsenic in the early part of his stay. The treatment he is mainly under now is a preparation of bone-marrow; but though he has improved in some respects, he is decidedly worse in others.

N.B.—Shortly after this lecture was delivered, the patient died of pneumonia. The following conditions were found:—Cervical glands enlarged and hard; spleen hard, not enlarged, free from deposit; liver enlarged, with whitish streaks of lymphoid tissue throughout it; pancreas thick and

hard, with a separate yellowish tumour in its substance; small round growths in the submucous tissue of the stomach; hard and enlarged salivary glands; the appendix cæci very long, thickened, and fixed to hard inflammatory tissue near the kidney; kidneys in a condition of nephritis with haemorrhages. Such histological examinations as have been made are in favour of the lymphadenomatous nature of the case.

A CONSULTATION IN THE OPERATING THEATRE OF ST. BARTHOLOMEW'S HOSPITAL.

Treatment of tuberculous ulcers in a myxœdematous patient.

Mr. Lockwood:—About two months ago Mr. Butlin had this patient under his care. She was suffering from a curious condition of her legs. A very large area of each was covered with granulomatous growth of a doubtful nature, but which was stated to be tubercular. At St. Mary's Hospital the tissue was examined, and was held to be tubercular. At the same time she had myxœdema, and was treated here for that complaint, and afterwards came under my care. I removed the granulomatous tissue from both legs, and you see that they are now healed. Healing took place very quickly indeed. She had also small doses of thyroid tablets. It would be difficult to recognise her again as the patient from whom we had removed the granulomatous tissue. Her mental condition, too, is very much improved. She was taking thyroid extract when the granulomatous tissue was removed. Dr. Kanthack now reports that the tissue was typically tuberculous.

Mr. Bowlby:—I congratulate Mr. Lockwood on the treatment, the patient's general appearance shows a marked improvement on what I remember.

Mr. Lockwood:—Her husband is one of the persons most struck with the change, and he says that now she is a very talkative woman, whereas before she was quiet.

Tuberculous disease of the bladder.

Mr. Lockwood:—This patient now brought in is under the care of Mr. Butlin. With the

cystoscope we saw clearly that he had tuberculous ulcers in his bladder. The bladder was opened, but the ulcers were so extensive that no scraping or anything at all was done. A tube was placed in the bladder above the pubes, and the bladder was afterwards washed out with iodoform emulsion. Mr. Butlin thought it ought to be shewn, so that the result of opening the bladder of a tuberculous patient should be seen, the wound having now become tuberculous. Mr. Butlin also wishes to know whether any further treatment would be recommended by his colleagues. I ought to add that Mr. Butlin's diagnosis of the tuberculous nature of this affection of the bladder does not rest entirely on clinical evidence; it has been examined microscopically and found to be definitely tuberculous. The operation has relieved this man's pain. Very severe pains were complained of by the patient before the operation. When the man's bladder was opened, only an ulcer was seen near the neck, but unfortunately the disease was more extensive than was supposed before the operation. I should not myself think, from what I saw, that anything more could be done. His fistula I am afraid will not close.

Mr. Langton:—Tuberculous disease may be seen to have spread to the wound. If a month ago this man's condition was not amenable to local treatment, it probably would not now be more likely to improve. I do not recommend any further treatment.

Mr. Bowlby:—I have myself a patient in the hospital now who was admitted three months ago with haematuria and a doubtful diagnosis of tuberculous disease of the bladder. It was arranged that the bladder should be opened, and that, if possible, the tuberculous mass should be scraped away. That was done, but without much benefit, and there seems to be in that case a probability, amounting almost to a certainty, that he will have a fistula which will not heal. That ought to be considered when one is advising the opening of a bladder for any ulceration which is presumably tuberculous, for although the pain and frequent micturition may be removed, a fistulous opening which will not heal may be the result.

Amputation of the Leg.

Mr. Marsh: I should be glad of the opinion of my colleagues on the patient I now show. He is

an omnibus driver forty-eight years of age. Seven years ago he slipped off an omnibus and was dragged on the ground, and the integument of both legs was lacerated. The wounds healed after a year, and in 1890 he resumed his work. Though the wounds healed for a time, ulcers soon formed, and these have continued. You will see that he has now very extensive ulceration of both legs. The left leg presents a feature requiring special attention; the ulcer almost encircles the limb. It passes from the outer aspect to the inner, and there is only a narrow belt of skin left posteriorly. I am speaking about it freely in his presence, because he has himself requested that the limb should be amputated. It interferes with his occupation, and he has been idle for two years. The question arises, can this ulcer be healed? Possibly it might be healed by rest and extensive grafting, but the healing would not last. My opinion is that he had much better submit to amputation. He would be well very soon, and then he could work. He is only forty-eight, and has been laid up for two years. As to the right leg, the ulcer there would have a better chance of healing when an artificial limb had been supplied for the other side. He could then spare the right leg more than he can at present. While he is convalescing from this proposed amputation we might employ grafting by Thiersch's method, and rest, towards healing the ulcer on the right leg.

Mr. LANGTON: It might heal up, but it will be sure to break out again. If you attempted to put a Thiersch grafting on the ulcer of the left leg, there would be much oedema of the foot from the contracting scar tissue, and the limb would be useless. The worst of it is, that you may ultimately have to amputate the right leg as well, as it is in as almost an unsatisfactory condition as the left leg. It will thus probably require the same treatment at some future date.

Mr. WALSHAM: I should say, take the left leg off first, and wait for the necessity to arise for taking the other off.

Mr. BOWLBY: I agree that it would be better to remove the left limb first.

Mr. LOCKWOOD: I should operate on the bad leg first, the left, and I should suggest that the skin on the amputated limb should be used for the Thiersch grafting for the ulcer on the right leg.

DEFORMITY OF FEET AND TROPHIC CHANGES IN JOINTS AND BONES ASSOCIATED WITH ? PERIPHERAL NEURITIS.*

By FREDERIC EVE, F.R.C.S.,
Surgeon to, and Lecturer on Pathology at, the
London Hospital.

MR. PRESIDENT AND GENTLEMEN,—The specimens I am about to describe are, I believe, examples of distortion of the foot, with trophic changes in the joints and bones from peripheral neuritis, or some nervous affection--not locomotor ataxy--associated with changes in peripheral nerves. The class of diseases under discussion this evening form a common meeting-ground for the physician and the surgeon, and I have brought these specimens here in the hope that some of you may be able to refer to similar cases, or give me some additional information as to their nature.

Case 1.—The first specimen is from a patient I saw in the out-patient department of the London Hospital. Both feet presented all the appearances



of extreme equino varus (see photo), and there was ulceration on the outer sides and dorsum where they rested on the ground. The history of the case is as follows:—The patient was a man æt. 27, an umbrella maker. About ten years ago he noticed pain in the toes, followed by pain in the ankles, and soon after the deformity commenced. Gradually the patient got worse, and six years ago the outer sides of both feet got sore from pressure in walking. Up to the age of seventeen the patient was quite strong, and both his feet were

* Specimens shown at Hunterian Society, 22nd April, 1896.

straight. There was loss of sensation in the lower thirds of both legs, with analgesia. No evidence of locomotor ataxy. The muscles of the legs were wasted, the right calf measuring $11\frac{1}{2}$ inches, the left 11 inches, in circumference. The skin of the dorsum of the feet was dusky, glossy, and in parts the hair was long. There was slight muscular contraction with the interrupted current in extensors, more in right tibialis anticus than left. Very slight reaction to the same in calf muscles.

My colleague, Mr. McCarthy, under whom the patient was admitted, amputated both feet ; he kindly allowed me to examine the specimens, and make use of the case. I found a remarkable condition of the joints (specimens shown). In the ankle-joint, and the joints of the tarsus in both feet, the cartilages were almost entirely destroyed. In places the bone was exposed and slightly dense, but the cartilage had been replaced for the most part by fibrous tissue, which firmly united the opposing articular surfaces, rendering separation difficult. The articular surfaces were altered in adaptation to the altered relation of the bones, but there was no destruction. The muscles on the plantar surface of the feet were wasted, and so largely converted into fat that they could not be separated by dissection from the superabundant subcutaneous fat. The great toes were everted, and the little toes showed the claw or "Z-shaped" deformity.

This condition presents a great difference from that found in Charcot's disease, where the capsules are distended, the bones eroded—the tarsus feeling often like knuckle-bones in a bag. In this case the cartilages were destroyed, and their place taken by fibrous tissue, uniting the joints firmly. I have observed a similar condition to this in the finger-joints in a case of long-standing injury to the median nerve. The anterior and posterior tibial nerves in both legs were enlarged, and the microscopic examination showed marked sclerosis.

Case 2. Of the other case I can only show you a cast, and here we have also a curious deformity of the foot, associated with peripheral neuritis. The patient was a young man æt. 19, a barman. The changes in the foot were of recent origin. Before I saw him he had been in hospital three months before, with ulceration of the right foot. Now he came for ulceration in the left foot. There

was no evidence of locomotor ataxy ; the knee-jerks were present, the pupils reacted to light and were equal, the plantar reflex was increased. The sensibility to heat and cold in left foot was much diminished, although he could distinguish between heat and cold in it, but not in the right. There was marked anaesthesia, but he could distinguish a prick from a touch on the legs. Both feet showed much the same condition. In the first case described, there was extreme equino-varus, and in this case there was a spurious valgus. The arch was obliterated, the astragalus had fallen, the toes, especially the great, were everted, deformed, drawn up, or lost. Another remarkable feature was ulceration at the ends of several of the toes, with loss of the nails. There was an ulceration at the end of the second toe, and on the inner side of the great toe of the left foot. The phalanges of the great toe and of the second toe were found to be loose and necrosed on opening up the sinuses. Both toes were amputated.

This condition of the toes is very similar to that observed in the fingers, in cases of anaesthetic leprosy, where we have, of course, enormous sclerosis of the nerves, the result of the leprous bacillus acting as an irritant to the nerves. The cause of the neuritis in the case related could not be discovered. He had not had syphilis. Being a barman, perhaps it was alcoholic, although no history of intemperance could be obtained. Dr. Ralfe kindly examined the case, but could throw no further light on this point, and confirmed the diagnosis of peripheral neuritis. The patient stated that a brother and an uncle had a similar affection of their feet.

Both these cases present one point in common with some cases of locomotor ataxy, namely, changes in peripheral nerves. In a measure they support the view that the tropic changes in joints, bones, &c. in locomotor ataxy, are largely due to changes in the peripheral nerves.

Coryza.—

B. Ichthyol	1
Ether,	
Alcohol.....	aa 1
Distilled water	97
M. S. To be sprayed into the nose.	

(*Le Progrès Médical*).

ON THE TREATMENT OF SOME MEDICAL EMERGENCIES.

Read before the Preston Medico-Ethical Society.

BY

SEYMOUR TAYLOR, M.D., M.R.C.P.,

Senior Assistant-Physician to the West London Hospital.

GENTLEMEN,—When one is asked by any society of medical men to read an address, there are, so far at least as I am concerned, two difficult questions which present themselves. The first is, "What subject shall I select?" Then, having fixed upon a subject for your address, the second question looms large, and assumes greater importance than the first. It is "Can I do justice to the subject?"

I am afraid this last proposition will always be with me, for however simple a topic I may have selected for this address, I have the fear, which knowledge of my own want of power engenders, that I can in no sufficient way render the subject so attractive as it should be.

Nevertheless, recently my time has been occupied in studying the great manuals of medicine which have been published during the last twenty years, and the conviction has come to my mind that in the treatment of some diseases we have, on the one hand, stood still to our patients' disadvantage, whilst on the other hand, notwithstanding great strides in some directions, yet in some complaints our so-called improvements do not warrant such a term, and had we stood still it were perhaps also better for our clients.

So it occurred to me that it might not be unacceptable to you if I presented to you my views, based on hospital and private practice, on the treatment of some medical emergencies, such as are likely to occur to each and all of you in the course of your busy lives. Hence I have laid my private note-books under requisition, and I have referred to the treatment of emergencies to which I have been summoned in hospital and in consulting practice. And I am pleased to think, on taking view of my past records, that my advice has not been sought for in one special line of ailments, but that diseases of widely different characters and ætiology have been brought under my notice.

To begin with; then, let me take such a common

subject as Acute Peritonitis. In every case which I have seen, one predominant symptom has always appeared to me to require recognition, and that symptom is *shock*. From whatever cause acute peritonitis may originate, whether it be from gunshot wound, or from blows, or from some other traumatism, whether it be from a ruptured appendix, stomach, intestine, or other organ, or whether it arise from the effects of ptomaines and other toxic agents,—as I believe it may do,—from whichever of the many conditions of and injuries to a sensitive membrane this inflammation may arise, shock has always appeared to me to be the one symptom of paramount importance, and requiring all our skill and care.

This conviction was forced on my mind in my earlier days, when surgery was to me an attractive study. I was confirmed in my views by hearing Le Gros Clarke's admirable lectures on the subject of "Shock;" but I rather think that this opinion dates from my still earlier years at school, when my holidays were spent in shooting marbles at rats, birds, and other small game.

My sporting experience soon told me two things, viz. that if a rat or rabbit, for example, were struck on the head by a bullet, it more frequently than not had strength sufficient to enable it to struggle back to its lair before it died; but if struck on the belly, death was instantaneous and on the spot.

Hence, in applying this experience to practice, I hold that our first aim in peritonitis is to counteract or diminish the effects of shock. Opium is the recognised remedy, but I doubt if its efficacy is in the direction of lessening peristalsis. I think it saves life in the same way as a syringeful of morphia may save the life of a soldier who lies out all night with a mangled limb. Hot fomentations also are valuable adjuncts; but here again I am of opinion that their utility is greatest in lessening shock rather than in relieving pain.

Take another example, viz. the peritonitis which is rapidly set up by perforation of the stomach or of the intestine. What is the cause of death in such lesions? Obviously shock. And what is our treatment? It is necessary to at once open the belly cavity and sew up the visceral wound. Although I have no right to be unduly assertive in a surgical question, yet from what I have seen of surgical practice in such cases as we are considering, I emphatically assert, and I am convinced that

most surgeons will agree with me, that the only chance of life is afforded to our patient by operation at once when the rupture shall have been diagnosed, that the operation must be speedily performed, every minute being precious, and that we must save time by administering anaesthesia to as low degree as possible. The shock of cutting through the abdominal parietes is as nothing compared with the shock of irritant contents of stomach or bowel, spreading themselves over a highly sensitive membrane. And there is yet another point which medicine may suggest to surgery, which is, that in operation on the stomach and the higher regions of the abdomen, free drainage should be secured either by incisions in the hypogastric area, or even, as I have heard Mr. Watson Cheyne has done, into the rectum. The tendency of the productions of inflammation is to gravitate towards the pelvis, no matter how you may arrange the patient's position in bed, and I am sure that I have seen more than one death occur from the effects of peritonitis lurking around the pelvic organs even after a successful mending of a ruptured viscus higher up.

To sum up, I think we should regard the peritoneum not only as a large absorptive membrane, but as an extensive area of nerve endings.

My next subject for consideration is *Hyperpyrexia*. Of late years it has become fashionable to ignore the use of drugs in this condition. The argument has been urged that few medicines have any marked power in reducing temperatures unless the remedies are pushed to such extremes in dosage as to render them a source of danger. But is this really the case? I admit at the onset that I prefer the use of ice packs or the graduated bath. They are quicker in their action, they are easily controlled, and very few houses are without the means of applying either one or the other. But have we no drugs upon which we can depend when we wish to bring a highly feverish and therefore perilous temperature down to a region of safety? I will leave on one side quinine and the phenol compounds, since possibly they may come within the objectionable category which is marked as dangerous. But I will draw your attention for a few minutes to a common food, or drug if you will, which is somewhat neglected as a cooling agent. I refer to alcohol. Probably most of us prescribe it somewhat casually and carelessly, with

no instructions as to exact quantity and the periods at which it is to be taken. But I am as certainly convinced as a man can be, that when prescribed with intelligence, it is one of the most powerful remedial and restorative agents which we possess.

My thoughts were first drawn to the subject of alcohol as a heat-reducing agent by the refusal of a northern gamekeeper to take some whisky when we were exposed on a cold night; he simply said when I proffered him a flask, "No! It is too cold." These words sank deeply into my mind and caused me much anxious thought. And I am sure that the experience imparted to me that cold winter's night has borne good fruit. I need not burden you with a recital of a series of illustrative cases; but I may perhaps be allowed to relate a case in which I was called in consultation some little time back. The patient, a heavy and unwieldy woman, was suffering from typhoid fever in the third week. Her temperature was 105.6° , a point above the danger line, as I shall discuss with you shortly. She was quite insensible, and was to all appearances about to perish from hyperpyrexia. It was impossible from the surroundings of the patient to administer a graduated bath, or even an iced pack. So we decided on giving alcohol in large doses. Between nine o'clock at night and nine the next morning our patient was made to take twelve ounces of good brandy, an ounce every hour. On the morrow I found her temperature down to 100.5° ; she was sensible, and appeared astonished when she saw and recognised me as a strange doctor, but was of course quite oblivious to the fact that I had visited her only about twelve hours previously. From this date she made an uninterrupted and good recovery.

Again, I have seen cases of measles in young children in whom the temperature was kept up above the three-figure line for some days after the date when the end of the fever should have occurred. Stethoscopic and other physical examinations failed to recognise any sufficient cause for this protracted febrility. It was evidently due to some continued and increased metabolism as a sequel to the specific fever. Free doses of brandy were administered, and in two days the children were well. This experience is not limited to one, nor yet two cases. I have observed it, and profited by it, in a fairly large number of cases not

only of measles but of whooping cough and other specific fevers.

Then you will ask, "Do not the graduated bath or the cold pack meet with your approval?" Yes, certainly they do. I think that the cutaneous surface is the quickest and most certain channel by which heat may be abstracted from the body. I cannot in a short, and as I hope practical, paper discuss the actions of cold ablutions, since they are as yet wrapt in mystery, or at least somewhat obscure. But I can safely say that you must pick your cases if you wish to administer a cold bath or an ice-pack with success. Your cases should be free from pulmonary congestion; they should have a vigorous heart action,—in short, it will not do to administer these remedies unless the pulmonary circulation is free and unimpeded. Here, again, this didactic assertion is the offshoot of practical observation.

Some years back, when I was, with my esteemed teacher, Dr. Ord, making careful analyses of the autumnal epidemics of enteric fever, I had occasion to make, or be present at, several autopsies on patients who had died after the graduated bath.

In those whose pulmonary congestions during life had been very pronounced, I observed post-mortem a marked collapse of lungs, these organs being limp, airless, and slatey coloured. Dr. Bristowe, who lost one or two such cases, was also much struck by these appearances of the lungs, and expressed an order to his house physician that he was not to bathe any more typhoid cases. About two weeks afterwards another of Dr. Bristowe's cases died, and the same collapsed condition of lungs were noted. Thereupon Dr. Bristowe thought that his condemnation of the bath was unjust, and he remarked that he should have attributed the state of the lungs to the action of the cold baths, had he not given orders for their discontinuance. It was not till we returned to the wards that he was informed that his instructions had, owing to a change in his house physician, not been carried out.

But although I ask you to be circumspect in ordering cold ablutions, I am also convinced that such remedies are of the greatest value in hyperpyrexia in cases where the pulmonary tract is free and healthy.

From this it is an easy step to ask your attention to some points in the treatment of Enteric Fever.

Naturally, after just considering hyperpyrexia, the first thing which comes to my mind is the subject of temperatures. It will not be necessary for me to excuse myself for introducing diagnosis, since to know a disease well is a long step towards its successful treatment. But it has occurred to me on several occasions to be called in to a case of enteric fever in which the diagnosis was difficult or doubtful. Many of you must have seen cases in which an irregular temperature occurs for ten days or a fortnight, during which time no diagnosis has been made. There is no excuse for want of diagnosis if the temperature is remittent only, but the difficulty is considerable when, as not infrequently happens, the temperature is actually intermittent, for say seven days, the thermometer showing a record about 100° in the evening and 98·6° in the morning. Subsequently the temperature may become remittent, and the diagnosis then becomes more easy. A good rule to remember is this. A febrile temperature with any marked morning remissions for fourteen days, and accompanied by no physical signs of inflammation of any organ or tissue to account for it, is most probably due to enteric fever. And so long as any doubt exists, insist on your patient remaining in bed, and being placed on a rigidly febrile dietary. I am pretty sure many lives are sacrificed in cases of typhoid, which, mild at first, become virulent subsequently, owing to their non-recognition and therefore improper feeding. Let me impress it on you this way. I find from my hospital notes that a considerably higher rate of mortality (about 5 per cent. more) occurs in those cases who come to the out-patient rooms with a history of perhaps ten days of fever, and with no restriction as to work or dietary during that period.

If I now pass on to treatment of an ordinary case, it may interest you to know the results of my own practice and that of others. A friend of mine once remarked to me that cases of typhoid in a hospital appeared to him to differ in many respects from the ordinary type, *i. e.* private practice. I had noted the same. In a hospital the cases seem to have either a marked severity in diarrhoea, or an obstinate constipation; whilst in private practice, irregularity of bowels, or alternations of diarrhoea with constipation, were most frequent. I think the explanation is not far to seek. The dietary of a hospital case is strict and rigid, milk preponderating

over any other article of food. Hence if diarrhoea continue after admission for any length of period, it is an indication of the severity of the fever, and probably of the number and depth of the ulcers. The temperature charts of such cases support this view. On the other hand, constipation, again, is a hospital symptom, and is, I take it, due to an almost exclusive milk diet, which is rigidly adhered to, although the attack may be a mild one. The irregularity of the action of the bowels which occurs in private practice, I have attributed to the less rigid dietary discipline which obtains in a private house, notwithstanding that your nurse may be most loyal in her endeavour to carry out your orders.

This matter of feeding leads up to a most important matter, viz. relapses. In a given number of cases of enteric fever a certain percentage of relapses will occur even if our diet-sheet be unimpeachable. But there are accidents of the disease, and errors of diet especially towards convalescence, which may lead up to and actually produce a relapse or a recrudescence. In the St. Thomas's epidemics we found a large percentage, indeed the majority, of relapses dated from either an action of the bowels after a period of constipation or from the allowance of bread in the dietary. No matter what the remedy employed to relieve the constipation, whether it was castor oil or a simple enema, the risk was just the same—a rise of temperature dated from the evening of the day on which the bowels acted. The return of febrility might last two or three days, amounting to a recrudescence only, or it might constitute a fresh attack or true relapse. Only last autumn I saw two in which I was called to consult as to the cause of such cases, a sudden return of fever. In both cases enemata had been given, and I was enabled to point out that the condition might be temporary only (of two or three days' duration), or it might develop into a genuine relapse.

But bread is a far more fatal error. We learned this from the nursing sisters. I cannot satisfactorily explain why it should be, but the most alarming and fatal relapses which I have observed were those which commenced on the day following an allowance of bread, whether it was given boiled with milk, or even as toast in the beef-tea. I will only quote one case. An old friend of mine was attacked with typhoid, and I saw him in consulta-

tion with his family doctor. Then I went away for a three weeks' holiday, being kept informed of his progress every other day. Towards the end of the disease I wrote to his doctor to ask him to exclude bread from his diet for at least another ten days. Three days after my return home I was summoned to his bedside, and found all the symptoms of a well-marked relapse. The second fever was more severe than the first, and he unhappily perished. Unfortunately, by some mistake a basin of bread and milk had been given to him on the day prior to the fresh rise of temperature.

As regards drugs, I have no rule of thumb to lay down. But my practice has followed on Dr. John Harley's plan,—to give small doses of grey powder three times a day. Whether the mercurial preparation acts as a partial antiseptic on the bowel, or whether by keeping up a gentle peristalsis and flow through the intestine, it obviates constipation on one hand, and diarrhoea on the other, I cannot quite satisfy my mind. I can only tell you of results. Dr. Harley's death rate in hospital practice was extremely low, and I have had similar happy results by following in his footsteps.

Then there is another experience which my notebooks have afforded me. A high temperature is always in itself a dangerous symptom. Wunderlich fixed the line of danger in typhoid at 105.5° , I think. I would place it even lower, at 105° . Those of you who meet with much typhoid take note of it, and see how many cases recover which at any time present a temperature of 105° or upwards. I do not say that recovery does not take place. I have known many cases get well after this high reading; but the general rule may be stated from a large number of statistics, that a patient who has at any time had a fever of 105° , either during the primary attack, or it may be during a relapse, eventually dies.

One other point, and I have done so far as enteric fever is concerned. We have a remedy, which as a stimulant is an exceedingly valuable, if neglected, one. I refer to musk. I formerly regarded musk as a disgusting remnant of a barbaric pharmacopoeia. But I was induced to try it in a case where profound exhaustion with subsultus were present, and when alcoholic stimulant was refused. The result was a happy recovery, and I have had reason to think highly of the preparation in some three or four subsequent cases.

I would next invite your attention for a few minutes to the consideration of *Acute Pneumonia*. As you all know, this is a disease which is for the most part sudden in its onset, and perilous to life, so that its treatment is one short chapter of terrible anxiety to us, and makes one desirous of asking a brother practitioner to share responsibility. Now, from whatever point of view one regards the disorder, whether from its symptoms, its fixed duration, terminating in a crisis, or its pathology, one must, I apprehend, favour the growing opinion that croupous pneumonia is a specific disease. Micro-organisms of well-recognised type (pneumococci) have been discovered; the disease runs a recognised course, so that we may safely say that on or about a certain day the fever will depart; the crisis is marked by some nerve-storm, whether it be delirium, or sweat, or diarrhoea; and finally Dr. Ord has had success in the treatment of the fever by giving large doses of tincture of iron, regarding the disease as akin to an erysipelas attacking the pulmonary tissue. My interest in the disease was awakened by the evidence afforded to me by a patient who was a stock-breeder in South Africa. He had, to his advantage, made some study of diseases affecting bovine animals, and told me that epidemics of pneumonia, and subsequently of pleurisy, often invaded their herds. I apprehend that the disease was epidemic pleuro-pneumonia, although he distinctly said that it was by no means always that "the pneumonia went on to water on the lung." But he told me of a rough-and-ready treatment somewhat anticipating the serum-therapeutics of to-day, which consisted of administering the pleuritic serum of an affected beast to such of his stock as showed no signs of the fever. He told me that all the cattle so treated had feverish symptoms for a few days, but that the mortality amongst the herd was reduced to a third of the previous death-rate. Modern research by experiment favours the opinion that in a few years, it may be sooner, we shall have a modified serum, a toxin, call it what you will, which shall rob this terrible disease of its perils. I need not quote authorities to you on this subject, since you probably are cognisant of certain articles which have appeared in the *British Medical Journal*, and other professional papers. But pending any definite and well-established inoculation treatment

on the lines I have mentioned to you, it is our duty to discuss appropriate treatment, and adopt such measures as experience tells us have been successful. Now, I am strongly convinced that an old-fashioned and now discredited treatment may be partially revived, in the treatment of pneumonia at least, with advantage. I refer to venesection. I have now prescribed this treatment in five cases with the best results. The pulse diminished in frequency, and became of better tone, the breathing became less rapid, and the crises arrived on the fifth day, and were less severe than is usual. I do not advise you to bleed every patient who has pneumonia; but I emphatically assert that it is a treatment of the greatest advantage in strong, plethoric robust men,—the type of man, indeed, who appears prone to this disease, and who succumbs to it in heavy proportion.

From pneumonia it is an easy step to such a subject as *Hæmoptysis*, and as it is a symptom on the treatment of which I may be supposed to entertain views which are not orthodox, I venture to ask your attention for a few minutes.

Doubtless many of you have been summoned more than once to the bedside of a patient who is suffering from severe haemorrhage from the lung; and if so, I venture to assert that you have had to play a double part. You have had to soothe and relieve the patient, and you have also had to allay the panic which existed in the house, and which reacted upon and excited the patient, to his great hurt. Here I would ask, and it is a point of great importance in immediate prognosis, "Did you ever see a patient die of lung haemorrhage?" I have in my experience at a large special hospital for diseases of the chest seen one death only. The vessel when ruptured is often retracted into the deeper tissues, and so, as it were, it controls its own haemorrhage. Well, if you will admit this, we have it at once in our power to stay the distress of the household. We can assure them that the danger is more apparent than real, or that the risk to life is more remote than immediate; and hence, by enlisting the co-operation of the invalid's family, we can easily clear the room of superfluous helpers, we can open a window to ensure a plentiful supply of fresh air, and we soon notice a beneficial change in the patient's condition. He becomes less excited and more restful, his pulse falls to a lower rate, and dicrotism is less marked, and he is then

in a better condition to respond to our medicinal remedies.

Now I have found, on reference to our many text-books on medicine, that an old, almost stereotyped method of drugging is still largely advocated. Such drugs as turpentine, gallic, tannic, and sulphuric acids, also certain preparations of lead, are still extolled in the treatment of pulmonary haemorrhage. But do you really believe in their efficacy in this condition? So far as my experience goes they are for the most part useless, and may even do harm. Suppose, for example, we give tannin, by which the albuminous matter surrounding the intestinal vessels is coagulated, or suppose we prescribe sulphuric acid, by which watery exudation from the vessels is retarded, or by whatever method these astringents act, their final effect is the same; not only do we produce a lessened action on the bowels, but we at the same time, and thereby, bring about an increased tension on the vascular system in general, and so favour a continuance of haemorrhage rather than stop it. I protest I would rather prescribe a saline purgative. And even if these intestinal constringents do exert an effect on so remote a region as a lung's apex, it can only be partial and evanescent. As a brother practitioner once put it to me: "It certainly does seem a long way round for the action of such drugs." To me a much more rational and scientific treatment is to give opium, the drug *par excellence*, which calms the nervous system, combined with digitalis. By these measures we speedily notice amelioration of symptoms. The patient is quieted, his pulse becomes better in tone, and cough, one of the conditions we wish to avoid, is lessened. Then as a last resource, we can fall back on ergot. All these drugs can be easily given by the mouth, but in these days of pharmaceutical advance and refinement, it is at times advisable, owing to the rapid way they can be administered, to inject the remedies subcutaneously. We all of us carry hypodermic syringes about with us, and tabloids of compressed drugs take up no room in our pockets. I would ask those of you whom I may convince, to try and note the effects of the above line of treatment. At any rate you have used remedies which act directly on the circulatory system, which you desire to have somewhat under your control.

As a last criticism on the subject of pulmonary

bleeding, I would discountenance the use of an ice-bag. For me at any rate it is difficult to recognise any beneficial action from such treatment. I fail to grasp the notion that any good can accrue from the action of ice through the chest parietes, (with its skin, its muscles, and its bones,) upon a summit of a lung which is not necessarily adherent to these parietes, and in which therefore the circulation is derived from an entirely different set of vessels. Further, it is hardly necessary for me to point to the discomfort to the patient which results from the application of a cold, heavy, pad on a part in which tenderness is already a pronounced sign.

I now pass on to another subject. I have in the last three years been called in consultation to four cases of whooping-cough complicated by convulsions. The first case luckily enlightened me. Eclampsia was the serious condition for which extra help was sought. Immediately after my arrival the child had a severe attack of cough, but without any inspiratory "crow." This, however, was followed by convulsion. The diagnosis which we arrived at was "whooping cough in its first stage," and that the convulsions were due to and consequent on cerebral engorgement from the cough. This view was confirmed three days afterwards, when the typical "crow" was heard, and the child developed symmetrical black eyes from coughing. The patient's younger sister subsequently developed whooping-cough, and being therefore allowed to occupy the same room, she, at my request, spent some time in cultivating a budding artistic ability, and made a sketch of her sister's face. Now, the bearing of all this on our treatment will at first appear obscure. But to me the first indication was to allay the cough. We are taught that children, especially infants, bear opium and its derivatives badly. I must demur to this. One has only to give the proper dose, and children tolerate it as well as adults. In this case, to the astonishment of my friend in attendance, I suggested morphia, and it acted like magic. I gave $\frac{1}{6}$ grain every four hours, with instructions to watch its effects, and to discontinue it if the pupil signs became very pronounced. The paroxysms were less frequent, they were less prolonged, there were no more convulsions, and the child made a rapid recovery.

Subsequently three other cases were treated on

similar lines with equally good results. I can claim no originality in this treatment. Henoch advocates it; but I doubt whether such apparently heterodox medicine is so well known as it deserves.

I will now consider my last question, lest by prolongation of my cavillings at recognised treatments I should become wearisome. I have, however, often been struck by the totally different lines of treatment which one reads of in text-books, and which one actually sees in practice. This remark especially applies to apoplexy. I can best put it shortly and tersely to you by saying that if one follows *book* treatment, we shall probably retain our connection with the patient's friends, but we shall no doubt do harm to the patient himself. On the other hand, if we only adopt such measures as are best calculated to do the sufferer no harm and possibly some good, we shall be accused by his friends of standing idly by and doing nothing. This they will resent, and probably send for the nearest practitioner to supplement you.

In other words, if we bleed, if we blister the nape, if we apply sinapisms to the calves, we only do harm to our patient, although his friends may thank us for our assiduity. The wisest and most humane treatment is that which we see carried out in a hospital ward, viz. absolute rest, supplemented by, it may be, a calomel purge.

Let me, whilst on the subject of cerebral haemorrhage, endeavour to enlist your concurrence with my dogma about the cerebral circulation.

It is commonly supposed, and indeed generally taught, that the cerebral arteries are tortuous, with a view to lessen the impulse of the blood current, and so arrest shock to the cerebral nervous system. This idea is not borne out by anatomical research. A long experience as a teacher in the dissecting room has taught me that tortuosity in an artery is invariably associated with movement, or, what is a modification of it, enlargement of the part or organ to which it is distributed. Let me quote examples. The facial artery is tortuous, to allow of freedom of movement of lips and other parts of the face; the lingual artery is tortuous, to permit of elongation of the tongue to, perhaps, twice its length when at rest; the vertebral artery is serpentine at the axis and atlas, so as to permit rotation and other movements at the occiput; the spermatic, the uterine, the splenic, the gastric arteries are all tortuous, in order to accommodate themselves to

the varying positions and sizes of the organs which they supply.

And the evidence on the other side of the question is quite as conclusive. The aorta after its arch is a straight tube; the renal vessels run at right angles to it, for there is no great movement in the dorsal spine, nor is there any in the kidney.

Nay, the cerebral vessels themselves are not remarkable for their tortuosity. The most important cerebral artery merely curves along the Sylvian fissure, and I know of no straighter vessel in our whole system than the basilar. Think of this, you who are interested in anatomy, and see if my assertions be not correct.

Gentlemen, I have finished. I fear I have told you little that is new to you, and much that is commonplace. I fear also that I may have been didactic at times and over-confident always, and that therefore I have perhaps upset some of your most cherished notions, and, metaphorically, trodden on some pet theoretical corns. But my intentions have been honest, and I have not based this address on any wild fancies of a brain fertile in imagination. I have merely given you the result of my observations and experience as recorded in my note-books. If I have made you view your cases from any different standpoint, if I have made you think of them with still greater breadth of mind than you already possess, I shall have been gratified, and the object of my address will have been attained.

REVIEW.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers. Small 8vo., price 4d. London. SIR ISAAC PITMAN & SONS, 1, Amen Corner, E.C.

A very readable paper on the Hæmoptysis of Phthisis, by Dr. Wethered of the Brompton Consumption Hospital, with a short note by Dr. Gowers on the use of the ophthalmoscope as an otoscope, comprise the original communications in the March number. Dr. Wethered divides cases of hæmoptysis into those due to aneurismal dilat-

tation of the pulmonary vessels (usually found in connection with cavities), to erosion of the vessels, to a morbid condition of the vessels themselves, and to active hyperæmia. We think, however, that his estimate that 60 per cent. of the cases of phthisis spit up as much as or more than an ounce of blood, is unduly high. Early haemoptysis is often a blessing in disguise, by drawing attention to latent and perhaps unsuspected disease, though blood retained in the lung may start fresh foci of inflammation. Haemoptysis when it kills, does so by suffocation. Hence the patient should be boldly encouraged to cough up the blood. The sight of the blood, and fear of impending death, paralyse his efforts, and the physician can do much to inspire confidence and allay fear. Too exhaustive an examination of the chest should not be made, and it is of the utmost importance to restore confidence among those about the patient. Small quantities of ice should be given, and though drugs are of little value, gallic acid in twenty-grain doses seems the most likely to do good. Ice applied to the chest is useless and harmful, and Dr. Wethered altogether ignores any reflex power it may have of contracting internal blood-vessels, similar to that of the cold key put down a patient's back to check an epistaxis.

Morphia in quarter-grain dose hypodermically is of all remedies the most useful in giving the patient the rest and quiet necessary to his recovery. All food must be taken cold, and the patient must stay in bed till the haemoptysis has quite ceased. In slight cases Dr. Wethered recommends—

Tinct. Hamamelis	3xv,
Acid Sulphuric dil.	m <i>v</i> ,
Aq.

Ter die sumend.

Cases of slight haemoptysis do well at Davos, but where hemorrhage is severe, home is the best place for them.

The rest of the journal is taken up with notes of the work of the Society of Medical Phonographers, and we note the projected petition to the General Medical Council, praying that shorthand may be made one of the optional subjects of the Preliminary Examination. If students knew the utility of this time-saving art, none would begin medical study ignorant of it. Its inclusion in the syllabus of the Preliminary Examination would doubtless act as an incentive to its acquirement.

THERAPEUTICAL NOTES.

Therapy of Acromegaly with Extract of the Pituitary Gland.—Marinesco (*Semaine Médicale*) reports three cases of acromegaly treated with glandula pituitaria. Two cases were of the "massive" type, a woman of 53 years and a man of 54), and one case (a woman of over 30 years) of the "giant" type. Under the treatment the severe cephalic pains were diminished in the cases of "massive" type, but the remedy had no effect upon the neuralgic pains of the limbs. The general condition improved, but Marinesco was unable to produce the slightest diminution of the hypertrophied members. Increased diuresis was the most conspicuous effect of the treatment. Marinesco and Marie believe that acromegaly depends upon a functional disturbance of the pituitary gland, but reject the hypothesis of Tamburini and Massalongo, that the hypertrophy is a result of hypersecretion of the gland. In occasional cases section has shown that the gland had suffered a change, and that the cells had been supplanted by elements of a different kind, which had not the power to supply the normal glandular secretion.

Unusual Variety of Hæmaturia in a Case of Vesical Tumour.—Dr. Ferria has observed the case of a patient, æt. 24, who complained of painful micturition and hæmaturia. This latter symptom had been two years in existence, haemorrhage occurring at considerable intervals at first; latterly more often, with increased pain and frequent urgent desire to empty the bladder. The haemorrhage occurred more abundantly at the close of the act of micturition till shortly before advice was sought, when severe pain occurred at the beginning of the act and bleeding on almost every occasion, and only at the beginning also, the remaining urine being limpid. On examination it was determined that there was a considerable degree of retention, and that while the prostate and seminal vessels seemed normal, the sound, when introduced, on approaching the neck of the bladder came in contact with a surface apparently irregular and friable. On relieving the retention the catheter enabled Dr. Ferria to recognize a tumour in the bladder. Suprapubic cystotomy was performed, and a papilloma found on the anterior bladder wall to the left side, about three finger breadths from the urethral orifice. The pedicle was slender, 2 and $\frac{1}{2}$ centimetres in length, and the tumour was of the size of half a walnut, with a prolongation on one side which reached the neck of the bladder and penetrated the prostatic urethra.

(*Annales des Maladies des Organes Genito-urinaires.*)

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 20, 1896.

MENINGITIS IN ITS SURGICAL ASPECTS.

The Inaugural Lecture delivered at
The Victoria Hospital for Children, 14th May, 1896,

BY
D'ARCY POWER, M.A., Oxon., F.R.C.S., Eng.
Surgeon to the Victoria Hospital for Children, Chelsea.

After a few preliminary remarks upon the object of the course of lectures, and a hearty welcome to those who were in attendance, Mr. D'Arcy Power said :—

GENTLEMEN,—It would have appeared presumptuous a few years ago if a surgeon should have spoken to you upon a subject which was then considered to be so strictly medical as meningitis. Apart from the fact that I agree most thoroughly with the dictum of John Read, who wrote in 1588 that “in these our daies, chirurgerie is deuided from phisick, not without great hurt to mankinde.”

. . . And I doe withall affirme that all chirurgians ought to bee seene in phisicke,” there remains the more substantial reason that cases of meningitis in this hospital are often treated jointly by the physician and by the surgeon. The cases are admitted under the care of my colleague Dr. Dawtrey Drewitt, and if, as sometimes happens, they recover, well and good, but if they go from bad to worse, as too often occurs, they are handed over to me that I may ascertain the results of surgical interference. It is thus that I have had experience in the treatment of these cases, and that I feel myself competent to address you this afternoon upon so difficult and unsatisfactory a subject as that of meningitis.

When a child is attacked with meningitis, the first idea which presents itself to the mind of the medical attendant is that the inflammation is of necessity tuberculous in origin. This error is due to the division of meningitis into tuberculous, non-tuberculous, and purulent forms. A classification, which is a good example of what is termed by logi-

cians “cross-division.” It is as misleading as that old classification of animals into the vertebrata and the invertebrata, which led us to believe that all the vertebrata were as important as all the invertebrata put together, though we had still to learn that they were merely a single sub-kingdom of no greater classificatory importance than any of the six sub-kingdoms termed collectively the invertebrata. So it is with meningitis, for though tubercle is a common cause of the disease, it is no more correct to think of it as the sole cause than it would be to consider every pneumonia, or every inflammation of bone, as tuberculous. Yet so firmly is this idea implanted in the mind that I have seen a really skilled pathologist examine the green, tough, and peculiarly smelling lymph associated with the presence of the pneumococcus, and express surprise that it was unlike what he had seen in other cases of meningitis; but he well knew the pneumococcic form, and would have recognised it had he seen the lymph anywhere else than at the base of the brain, where his preconceived ideas had blinded him as to its origin.

The correct classification of meningitis is based upon pathological data. It is either infective or non-infective. The infective forms are the tuberculous, the pneumonic, the influenzal, the suppurative, and perhaps, to make the tale complete, the typhoidal and the malarial. Of these the tuberculous is the form with which we are the best acquainted in children, and it is usually, but by no means necessarily, the commonest. The simple or non-infective forms are the passive or congenital, the traumatic, the reflex, and the hysterical.

Infective meningitis itself can be divided into two classes. The first comprises those cases in which the inflammation of the cerebral membrane is associated with the presence of micro-organisms,—tuberculous, pneumococcic, influenzal, or pyogenic. The second group is that in which no micro-organisms can be found, though the inflammation is clearly infective in origin. The symptoms in these cases must therefore be due to the products of their action, that is to say, to toxins circulating through the brain.

The symptoms of meningitis are so well known that I may be excused from describing them to you, or from doing more than warning you not to lay too much stress upon the absence of optic neuritis, upon non-retraction of the head, or upon the presence of a tumid abdomen. An inflamed fundus oculi, cervical opisthotonus, and a keeled belly are valuable signs of inflammation of the cerebral meninges, but their absence individually or collectively does not preclude a diagnosis of meningitis. I shall therefore invite your attention at once to the two points of the greatest practical importance, which are always the first to present themselves to every medical man when he is called to see a sick child. The two questions are first, "What is the matter with the patient, and what can be done for his relief?" and secondly, the question which is of far greater importance to the parents than any mere diagnosis, "Will the boy recover or will he die?"

A child may suffer from severe headache with constipation, vomiting, fever, delirium, convulsions, and drowsiness passing into coma, without any inflammation of the cerebral membranes. Such a series of symptoms may occur in the course of typhoid fever; but the rise of temperature is not then associated with squinting, unequal pupils, transient disturbances of the vaso-motor system as is shown by pallor alternating with flushing, nor will there be hemiplegia, changes in the respiratory rhythm, or alterations in the pulse bearing no relation to the changes in the temperature of the patient. Besides, in typhoid fever the patient is generally sluggish and apathetic, whilst in meningitis he is often unduly sensitive to external impressions, so that there is hyperesthesia, and the various reflexes are exaggerated, whilst in typhoid they remain normal. Still, the differential diagnosis between typhoid fever and meningitis often remains obscure until it is cleared up at the end of the week, when the belly becomes tumid and the characteristic rose rash appears upon it; but in difficult cases it is well to remember that typhoid fever is less common in very young children than is meningitis, though enteric fever, as Dr. Northrup has shown, occurs even in sucklings.

Pneumonia is perhaps more likely to be mistaken for meningitis than any other affection to which children are liable. This is not surprising when the diseases are compared either from a

clinical or from a pathological standpoint. Clinically, pneumonia in children often begins with a headache and with disturbed sleep, and there are either actual convulsions or convulsive twitchings and a subsultus tendinum more suggestive of cerebral than of pulmonary trouble. In pneumonia, too, there is great prostration, and vomiting is of frequent occurrence; but in a very short time pneumonia is characterised by a short dry cough, and by a rapid rise of temperature with a disproportionate quickening of the pulse. Physical examination will then determine the nature of the disease, for auscultation reveals that the respiratory murmur is less marked, whilst percussion shows the presence of an obscure dulness over certain parts of the chest. I need not dwell upon these points, however, for Dr. ~~Caro~~ to deal with them in a future lecture. We know, ~~too~~, that the same poison will produce meningitis in one child and pneumonia in another. ~~This is not a matter for surprise, for in both cases the disease is associated with micro-organisms which grow in connection with serous membranes, and there is no reason, so far as we know at present, to prevent such a micro-organism from settling in the cranial rather than in the thoracic cavity.~~ APR 2 1897 A pneumococcus has been actually found in the cerebro-spinal fluid in some cases of meningitis, but in other cases the fluid has been sterile, and in these it must be assumed that the symptoms of meningitis are the result of the general constitutional disturbance produced by the toxins formed by the micro-organisms in other parts of the body, and the same explanation must hold good for the meningitis occurring in the course of typhoid fever.

But enough of transcendental pathology, lest you be wearied, and think that you are wasting that part of your valuable time which you sacrificed to come here this afternoon. Let us continue our consideration of the conditions which may be associated with meningitis in a child. We have seen that it is possible to mistake typhoid fever and pneumonia for this condition; but it is equally possible to mistake any of the exanthemata, during the invasion period, for an attack of meningitis, and in these doubtful cases a very guarded diagnosis should be given until some decisive sign makes its appearance.

The recent epidemics of influenza through which we have passed, have been fruitful in their yield of abnormal cases. The disease has invariably found

out the weak spot in each patient's body, and it is some consolation to those of us who have sustained one or more attacks without serious sequelæ to reflect that, for the present at any rate, we have no particularly vulnerable tissue or organ. Joint lesions after influenza have been frequent, but cases marked by cerebral symptoms have been no less frequent, and amongst these, symptoms indicative of meningitis have been by no means uncommon. I have taken the trouble to examine the statistics of this Hospital to ascertain what effect, if any, the influenza years had upon the number of cases of meningitis admitted into our wards. The records extend from 1880 to 1895, and during these years our average annual number of children suffering from meningitis, serous as well as purulent, has been eight. In 1890, the year of the first outbreak of influenza, the number rose to 19, in 1891 it was 24, in 1892 it was 18, in 1893 it was 14, whilst in 1894 it had sunk to 10. We may fairly assume, therefore, that the influenza had a definite share in producing this rise in the number of cases of meningitis, though we were too careless or too ignorant to distinguish between the cases directly due to influenza, and those caused by other affections.

There remains yet another group of symptoms which may be mistaken for meningitis, in which various peripheral causes acting upon the vascular and nervous symptoms lead to a mental condition so closely allied to meningitis as to render it difficult to distinguish the one from the other. These cases may be grouped as irritative or reflex meningitis, though they are more probably due to congestion of the substance of the brain than of its membranes. In towns they occur in rapidly growing children with some degree of ametropia, who are overworked at school ; they occur, too, in the children of gouty, rheumatic, and headachy parents. They are common all round our coast during the summer holidays in children who, fresh from the restrictions of town life, have been allowed to paddle for long mornings in the sea with a blazing sun overhead, and they are then akin to heat apoplexy or a slight sunstroke. They are particularly difficult to diagnose when they occur in children with a markedly tuberculous family history.

It will be sufficient to mention that Dr. Ollivier at Marseilles has called attention to hysterical

symptoms in children which nearly simulate those of meningitis, for we are a little apt to overlook hysteria in very young children, though Charcot long since showed that even the youngest sometimes inherit and exhibit a marked neurotic tendency.

The slowly progressive changes leading to cerebral sclerosis in inherited syphilis are amongst the rarer conditions which may be mistaken for meningitis. They occur in quite young children, and are hardly distinguishable from true meningitis, for there is headache, wakefulness, and vomiting, with or without convulsions. The symptoms in syphilis disappear in a few days, but there may be a fresh attack a few weeks or a few months later, with cerebral vomiting, strabismus, unequal pupils, and the sharp cry which we are accustomed to associate with meningitis. It is the recurrence of these attacks which leads us to suspect their true nature, and in doubtful cases we should ask whether there has been a previous attack, or we may give an unfavorable prognosis, which the child, fortunately for itself, will falsify.

The various forms of meningitis and its allied conditions which so far have been brought under review have been associated with a serous effusion, but there remains another great group in which the cerebral effusion is purulent, and this constitutes suppurative meningitis. The disease may be primary and traumatic, or it may be, as is most often the case, secondary to a purulent inflammation of the middle ear. Sometimes it is tuberculous, and occasionally it is associated with the pneumococcus, for Dr. Netter has shown that the products of such a suppurative meningitis are capable of setting up a typical pneumonia when they are injected into the lungs of a guinea-pig, although the lungs of the original patient were quite free from disease. Time prevents me from doing more than mentioning suppurative meningitis ; but I may remind you that it often sets in with startling rapidity, that it is marked by the most agonising headache, which is fortunately relieved by coma, and that unless active surgical measures be taken at the onset the patient will assuredly die.

This brings us to the prognosis of meningitis. Tuberculous meningitis is fatal ; trephining and draining away the cerebro-spinal fluid reduces the intracranial pressure, and prolongs life for a period of days or even weeks, but with the very rarest

exceptions an attack of tuberculous meningitis terminates in death. Death is a frequent termination of the other forms of meningitis, but in them recovery is somewhat more frequent. A meningitis in which the symptoms appear suddenly in an otherwise healthy child, or in the course of an attack of pneumonia, influenza, or typhoid fever is more likely to recover than one in which the symptoms come on insidiously. These slightly more favorable cases are often characterised by violent oscillations of the temperature, so that the chart shows a rise or fall of several degrees in a few hours. The urine is free from albumen, but its earthy phosphates are in the proportion of one, two, or three of the alkaline phosphates instead of the equal parts which exist in health, whilst the urea and chlorides are reduced to nearly half their usual quantities. Our prognosis, however, in cases of meningitis is largely influenced by the temperature chart, for prolonged pyrexia is always of bad import.

What I have said will show that the diagnosis of meningitis is often one of extreme difficulty, and that when it has been recognised it is not always easy to determine the particular form of inflammation with which we have to deal. The difficulty of diagnosis and its importance is brought home to us, I think, when we read that dreadful history of the witch mania which affected so large a part of Europe in the xvith century, and the neighbourhood of Salem in North America in 1692. Many of the unfortunate victims who were first tortured, and then cruelly killed, were charged with "overlooking" children, who afterwards pined away and died, some rapidly within a few days, others after a lingering illness of weeks or months. Many of these children complained of pains in their heads, they became unconscious, were convulsed, and died. It is obvious from their histories that some of the children suffered either from general tuberculosis or from some form of meningitis. How much wanton cruelty would have been spared if our ancient brethren in the profession had been able to make a better diagnosis! We shall not fall under any shadow of this kind, yet an accurate knowledge of the nature of the disease is of the utmost importance, for it determines the prognosis even if it does not materially influence our treatment.

The differential diagnosis of meningitis has been empirical until quite lately, when the operation of

lumbar puncture of the subarachnoid space has enabled us to place it upon a rational basis. The operation is a trivial one, for no greater danger attends it than attends paracentesis of the chest or of the abdomen; yet because it involves the passage of a trocar into the theca vertebralis, practitioners shrink from performing it, and too often content themselves with a vague diagnosis of meningitis, which in cases of death is not always verified by an autopsy.

The operation consists in withdrawing cerebro-spinal fluid from the lowest part of the vertebral canal by means of a puncture carried through the ligaments connecting the lumbar vertebrae. It arose, as often happens with new methods, in two countries about the same time. Dr. Essex Wynter practised it in England as early as February, 1889, whilst Quincke published his results at the Tenth Medical Congress held at Wiesbaden in 1891. Both hoped that the operation might be curative. Wynter, indeed, performed it with the idea of draining away the cerebro-spinal fluid, thereby relieving the patient by diminishing the intracranial pressure in much the same manner as tracheotomy relieves the dyspnoea in diphtheria. Quincke demonstrated as early as 1872 that it is possible to inject fluid into the subarachnoid space by means of a hypodermic syringe, and that by injecting red sulphide of mercury it is easy to prove the existence of a free communication between the subarachnoid spaces of the brain and of the spinal cord. The first attempts were experimental, but Prof. von Ziemssen, Drs. Lichtheim, Fürbringer and Stadelmann in Germany, and Dr. Jacoby in New York, have devoted much attention to the operation, which by their endeavours is rapidly being placed upon a scientific basis.

There are many ways of performing the operation. The puncture may be made with a hollow needle, with a trocar and cannula, or with a hypodermic syringe, but it is better not to aspirate. Personally I perform the operation by placing the child upon its right side, with its thighs so bent that it lies curled up with the vertebral column well bowed. This is easy in comatose children, but it is often difficult in those who are suffering from cerebral irritation, in the restless, and in those who are not yet unconscious; for such children it is necessary to give an anaesthetic. The exact position of the patient is unimportant so long as the vertebral

column is kept convex at the seat of puncture from the time the needle is entered until it is withdrawn.

The skin over the lower part of the back is well washed with soap and water, dried and sponged with a solution of corrosive sublimate 1 in 2000, whilst the trocar and cannula, of the smallest hydrocele size (No. 1 of English instrument makers), is sterilised by boiling in a test-tube for three minutes. I then drop a perpendicular upon the bed from the highest point of the crest of the ilium, for this line crosses the upper border of the spine of the fourth lumbar vertebra, and thus marks the position for the puncture. This point is selected because in a child at birth the spinal cord only reaches as low as the third lumbar vertebra, whilst in the adult the lower limit of the cord is level with the lower border of the first lumbar vertebra. It is therefore perfectly safe to puncture the vertebral canal even in a young child between the third and fourth or the fourth and fifth lumbar vertebrae. Quincke, too, has shown that the cauda equina in children is less dense than it is in adults, and that it is generally divided into two bundles which lie upon either side of the vertebral canal, leaving a space of about a quarter of an inch occupied by cerebro-spinal fluid.

The place being thus selected, and the reason for its selection being fully understood, the trocar and cannula are plunged through the skin immediately to one side of the spine of the third lumbar vertebra and on a level with its lower border. It is pushed on boldly until the point of the trocar touches bone—the lower border of the lamina. The handle of the trocar is then directed upwards so that its point passes downwards over the lamina, and it is then again pushed onwards until a grating sensation is felt, caused by the trocar passing through the ligamentum subflavum. There is sometimes a little difficulty in making the cannula pass through the resistant ligamentum subflavum, unless its end is so fully bevelled that it fits closely to the neck of the trocar, and it is, therefore, as well to look to this point when the instrument is selected. The trocar is withdrawn as soon as it is felt that it has fairly entered the subarachnoid space, care being taken that the cannula is not at the same time pulled out of the vertebral canal, though it is not very easy to do so. In a typical case of meningitis with serous effusion,

clear fluid will issue from the cannula drop by drop, the rate of flow being greater when the patient is raised than when he is recumbent. Some of you will remember that in a patient to whom I shall refer in greater detail presently (Case 2), I withdrew more than 200 minims of limpid fluid without any admixture of blood in less than five minutes. The rate of flow in this case altered synchronously with the respiratory rhythm, for it was slow during inspiration, whilst each expiration caused it to issue almost in a stream—a variation which agrees with the clinical and experimental observations of Burrows, Ackerman, and Donders, who have shown that the brain and spinal cord contract during each inspiration and expand with each expiration. Every systole of the heart, too, is accompanied by an expansion of the brain and spinal cord, whilst each diastole is associated with their contraction, as may readily be seen, so far as the cord is concerned, during the performance of the operation of laminectomy.

I daresay that those of you who saw the case will also remember how marked was the fall of temperature after the removal of the fluid, but of this, too, I shall speak in greater detail presently. The flow of cerebro-spinal fluid was stopped because the child had a slight convulsion. Mr. Jones kindly examined what we had withdrawn, and found that it did not reduce a boiling and alkaline solution of copper sulphate, but that it became cloudy on boiling, and gave a marked ring with nitric acid in the cold. It contained a small conical clot, formed spontaneously after it had stood for twenty-four hours, and this clotting is said to occur more frequently in the cerebro-spinal fluid from basal meningitis than in that obtained from other forms of disease. Leichtheim says that in cases of cerebral tumour the fluid obtained by puncture contains less proteid matter and is therefore less liable to clot than that found in basal meningitis. It contains, too, more of the copper-reducing substance, for fluid from tuberculous meningitis rarely gives the sugar test. Mr. Jones also stained the fluid, and a careful search showed the presence of a few tubercle bacilli with a somewhat larger proportion of diplococci.

Micro-organisms are not always present in cerebro spinal fluid, and they are often so few in number that a diligent search is necessary before their presence can be affirmed or denied: nor is

cerebro-spinal fluid always clear. It may be turbid, pus has been withdrawn occasionally, and in one case of cerebral haemorrhage with rupture of the cerebral ventricles, and in another of haemorrhage into the fourth ventricle, blood issued from the spinal puncture.

The operation is "neither dreadful in the doing nor melancholy in the event," as Samuel Sharp, the great surgeon to Guy's Hospital in the middle of the last century, wrote of extirpation of the tonsils. There are, however, a few sources of failure to be enumerated, as well as a few precautions to be taken; thus the surgeon may fail to puncture the spinal dura mater through want of boldness, for the vertebral column lies at a considerable depth, and even in an emaciated child the muscles covering the laminæ may be an inch or more in thickness. It seems, therefore, as if he were going unnecessarily deep, but he cannot go wrong if he enter his instrument close to the side of the spine, and at its lower border. He may err on the side of over-boldness, and enter his trocar so clumsily that its point is embedded in the lamina. It may then be difficult to withdraw it, or if a needle be used its point may become so bent as to render it useless. When the spinal canal has been safely entered, there is still a danger that any sudden movement on the part of the patient may straighten the vertebral column, and may bring the laminæ so closely together as to prevent the withdrawal of the instrument; and it is conceivable that under such conditions a slender or too highly tempered needle may be snapped off short. The possibility of such an accident has always been present in my mind when it has been necessary to perform this operation, and I have therefore preferred to use a trocar and cannula, rather than a hypodermic syringe. There is also a danger of wounding the dorsi-spinal or meningo-rachidian plexuses of veins, but the danger is rather theoretical than real, for I have never seen any bleeding of importance after puncture. Dr. Jacoby says that he once drew off five cubic centimetres of pure dark blood instead of cerebro-spinal fluid. The blood undoubtedly came from the puncture of a spinal vein, for upon the second attempt he had no difficulty in getting clear fluid. If such an accident should happen it would be better to withdraw the needle, close the puncture with a piece of gauze soaked in collodion, and either enter the instrument upon the opposite

side, or wait until the following day before a fresh attempt is made, for the presence of blood in the fluid impairs its value for diagnostic purposes.

Very few bad effects have been recorded from the practice of spinal puncture in cases of meningitis. Pain has sometimes been felt, due perhaps to puncture of a nerve, and in a few cases there has been a spastic condition with some numbness of one leg, but the symptoms have soon passed off. They must in any case be rare, for Dr. Lebharz says that he has never seen any irritation, though he made 150 punctures in eighty-five cases. Fürbringer has called attention to a more serious result of spinal puncture, for in his record of eighty-six cases in which the operation was performed, he states that on five occasions death occurred within forty hours of the puncture. Three of these patients had intracranial tumours, whilst the other two were suffering from uræmia. He therefore warns surgeons not to puncture the subarachnoid space in cases of intracranial and especially of cerebellar tumours, for he thinks that there is abundant evidence to prove a causal connection between the puncture and death. Fortunately, however, there is reason to think that his experience is exceptional, for other surgeons have punctured in similar cases without any such disastrous results.

It is better to withdraw the instrument whilst the fluid is still dripping, to prevent any chance of germ-laden air passing into the spinal canal, though its entrance would probably not be a matter of any great importance. The removal of the cannula often requires a good sharp tug, as the ligaments hold it firmly. A small pad of gauze soaked in collodion is sufficient to close the puncture, and I do not consider it necessary to apply a bandage.

Much positive evidence of great value in the diagnosis of an extremely difficult class of cases can be obtained by puncturing the subarachnoid space in the lumbar region, but I do not think that it can in any way be looked upon as curative, though Dr. Rieken says that the beneficial effects of spinal puncture are greater in cases of serous than of tubercular meningitis, and that in two of his cases the improvement followed so immediately upon the withdrawal of the cerebro-spinal fluid as to leave no doubt in his mind that it was due to it. He believes that the removal of fluid in these cases, even though it does not save life, leads to an

improvement in the patient's condition, for it admits of a more rapid absorption of the remaining exudate by the veins and lymphatics.

Too much importance must not be laid upon the absence of fluid, even when the puncture is made by one who has had experience in the operation, and who is certain that he has entered the vertebral canal. It may be that he has failed to pierce the theca vertebralis; it may be that the fluid is too thick to pass out; it may be that there is no fluid in the subarachnoid space. In such cases he should not be content with a single puncture, nor should he base his diagnosis upon a single bacteriological examination of the fluid, for what appears to be sterile upon one occasion may be found to contain micro-organisms in abundance at another time. Dr. Rieken lays special stress upon the measurement of the pressure of the cerebro-spinal fluid, as he considers that it is pathological when it exceeds 150 mm. He further states that a moderate increase in pressure with severe symptoms points to an acute attack of inflammation, whilst a very marked increase in pressure attended with comparatively slight symptoms affords evidence of a more chronic affection. In other words, the pressure symptoms in disease of the brain and cord depend more upon the rapidity with which the pressure increases than upon the absolute degree of pressure exercised by the cerebro-spinal fluid. It is inconvenient in practice to measure the pressure accurately by means of a manometer, though it is said not to be difficult. It is, therefore, rather guessed at from the rate of flow, for the pressure is high when the fluid flows out in a continuous stream, increased when it issues rapidly drop by drop, and normal when only a few drops can be obtained. It should be borne in mind, however, that the free communication which occurs in health between the subarachnoid space of the brain and the spinal cord is sometimes cut off by disease, and in such cases the diagnostic value of spinal puncture is limited, for no indication can then be obtained of the amount of the intracranial pressure, or of the character of the intracranial fluid.

A second method of surgical procedure has been adopted upon rather an extensive scale in the wards of this hospital. It consists in trephining the skull for the purpose of diminishing the intracranial pressure in cases of meningitis. We have performed the operation in the cerebellar

fossa between the superior and inferior curved lines, as near the middle line as possible, for the bone is there thinnest, and we have removed a crown of bone from the side of the skull. The first method allows of free drainage of the subarachnoid space of the skull, the second permits the lateral ventricle to be drained. The operation depends for its efficacy, if not for its legitimacy, upon the following considerations. The symptoms in meningitis are due in part to pressure exercised upon the brain by the serous or other effusion produced within the skull, in part to the toxic effects produced by the growth of the micro-organisms, and in part to the effect of the destructive inflammatory changes upon the nervous or connective-tissue elements of the brain itself. These different factors enter into every case of meningitis, but they appear to vary with the particular form of the disease. The toxic effects seem to be best marked in the typhoidal and pneumonic forms of meningitis. The pressure effects in those cases of sterile and passive effusion to which we still assign the old name hydrocephalus, whilst the destructive changes are very advanced in the tuberculous form. Withdrawal of the fluid certainly acts by diminishing the intracranial pressure, though the fall may be only temporary. It may also act in a more permanent manner, for we know that micro-organisms are very difficult to cultivate, and that slight alterations in the chemical and physical conditions of the media in which they grow may lead to their destruction. This is particularly well seen in cases of tuberculous peritonitis with effusion, in which the mere opening of the peritoneal cavity is sometimes followed by a cure, even though nothing has been done except to let out the serous effusion. The removal of the fluid necessarily means the removal of a certain number of micro-organisms, and if no active focus of inflammation exists, no more toxins may be produced, for so few micro-organisms may remain that the tissues of the patient's body may be able to deal with them, and recovery from some of the less deadly forms of meningitis may be the result of the operation.

Experience has taught us that little good can be expected from trephining or puncturing in cases of tuberculous meningitis, even in cases where the disease is not part of a general tuberculosis. The life of the patient may be prolonged for a few days

or for a few weeks, but eventually he dies, whether an operation has been done or whether he has been left under the care of the physician. The recent and very valuable paper by Dr. Ludvig Hektoen, of Chicago, published in the first part of the *American Journal of Experimental Medicine*, gives us a clue to the constant failure of operative measures in these cases. He made it his business to ascertain what vascular changes took place in tuberculous meningitis, and for this purpose he made a careful bacteriological and histological examination of nine cases occurring in persons of all ages. Eight of the cases showed that tubercle bacilli alone were present, but in the ninth case there was a mixed infection of tubercle with staphylococcus. Cultures of tubercle were readily obtained by inoculating a drop or two of meningeal fluid upon glycerine, agar,

temperature in cases of meningitis is due to intracranial pressure, but many more cases are required to establish the thesis, and I only wish to draw your attention to the fact that you may watch and carefully record whether there are the same concomitant variations in those cases which come under your own observation. I regret now that I did not take the temperature on both sides of the body, for it would be interesting to know whether in these cases the fall in the body heat is or is not bilateral. It is certainly unilateral in some cases, for in the last case of meningitis I saw, the temperature in the right axilla was 100.6° F., and in the left 101.6° F. The cases were :

Case 1. A. C.—, æt. 6 months. The child was strong and healthy until it was seven weeks old. It then had a rigor, was convulsed, and remained

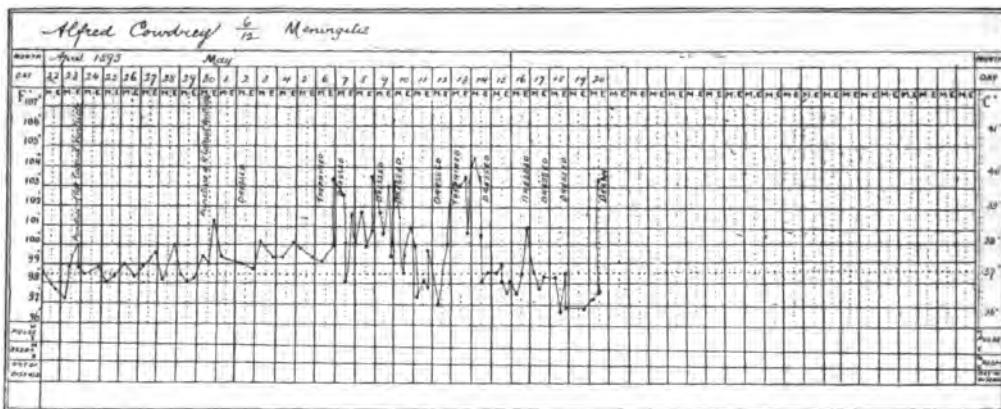


Fig. 1. Chart showing the fall of temperature following operations leading to a reduction of intracranial pressure.

or blood serum. The histological results were still more interesting, for in every one of the nine cases extensive vascular changes were present. Arterioles as well as arteries were involved, and it appeared as if any of the coats might become primarily diseased. The veins, too, were constantly the seat of a more or less extensive infiltration, but in them the change began in an extravascular or in an arterial focus. Dr. Hektoen believes that in some cases meningitis may be produced by infection from the blood itself, which brings the tubercle bacilli from other sources, deposits them in the smaller arteries, where they set up an endarteritis leading to caseation and hyaline degeneration of the whole wall.

Two cases which have come under my care recently appear to show that a portion of the increased

unconscious for three days. Its head had been enlarging slowly ever since the fit. The temperature of the child on April 23rd was 100° F. (Fig. 1.) It was anaesthetised, and by puncturing through the anterior fontanelle I drew off five ounces of blood-stained cerebro-spinal fluid from the left lateral ventricle. The pulse immediately improved in volume and in strength, whilst the temperature, as you will see by the chart, fell to 98.8° F., and did not again rise to 100° F. until April 28th, or five days later. I again punctured the brain on April 30th, directing the needle into the right ventricle; five ounces of cerebro-spinal fluid were removed, and although there was a rise of temperature immediately after the operation, it fell next morning to 99.4° F., and did not rise much above 100° F. until May 6th, when, as the child was restless, the

fontanelles and sutures were bulging, the head was retracted, and opisthotonus was becoming marked, I trephined the skull over the descending horn of the right lateral ventricle, and removed five ounces of clear cerebro-spinal fluid by pushing a trocar and cannula through the dura mater. Drainage of the ventricle was carried out by means of a dozen horsehairs bound together, and passed along the track of the cannula into the ventricle. The child had a little rigidity of the left side after the fluid had been drawn off, but it passed away before he was removed from the operating table. The temperature rose to $103\cdot4^{\circ}$ F. directly after the operation, and possibly as the result of the irritation produced by the presence of the horsehair drain passing through the brain substance, but it had fallen on the following morning to $98\cdot4^{\circ}$ F., and the ward note states that the child is distinctly improved. The symptoms of cerebral pressure again became pronounced on May 8th, when the temperature rose to $103\cdot4^{\circ}$ F. I dressed the wound, and found that the horsehair drain had nearly disappeared beneath the skin-flap, and that no cerebro-spinal fluid was escaping. The bundle was therefore readjusted, and I satisfied myself that it was acting efficiently as a drain before the dressings were re-applied. The ward note on the 10th, *i.e.* two days later, says, "The child has been better since the wound was dressed, and there has been so free a discharge of cerebro-spinal fluid that a daily dressing is necessary. The temperature is lower, there is less sickness, and the child takes food better. The wound too has healed, except at the place where the bundle of horsehair lies, and the sutures have therefore been removed. May 12th.—"There is still good drainage; the temperature is lower, and the general condition is distinctly better. There is less retraction of the head, and the opisthotonus is less marked. The child cries out less, and does not resent being disturbed so much." All the symptoms of intracranial pressure recurred upon the evening of May 12th, and the temperature then rose, until on the following morning it was again $103\cdot4^{\circ}$ F. No fluid had passed along the horsehair drain since the previous evening. I therefore opened up the subarachnoid space, about an inch and a half below the occipital protuberance, and as near the middle line as it was safe to go. A drainage-tube was passed into the cavity of the skull, and one end

was brought out through a hole in the centre of the flap. There was a free flow of cerebro-spinal fluid, and though the temperature fell that evening to $101\cdot6^{\circ}$ F., it rose during the night to $104\cdot6^{\circ}$ F. The cerebro-spinal fluid continued to escape, and on the following day the temperature fell to 98° F. It remained low for two days, whilst there was a free discharge of fluid, but on the 16th it rose to 101° F. The tube was explored with a probe, and by exercising a little gentle pressure upon the skull a free discharge of fluid was again obtained. The temperature fell immediately, and remained low until it rose again to $103\cdot4^{\circ}$ F., just before the child's death on the 20th.

The autopsy in this case showed that there was neither tubercle, syphilis, nor rickets. I examined the cerebro-spinal fluid carefully for micro-organisms, but I could find none. I injected some of it into the peritoneum of a guinea-pig, but there was no reaction, and the animal was alive and well six weeks afterwards. The cause of the condition was very obscure. The brain was thin and expanded; the subarachnoid space contained about two ounces of clear cerebro-spinal fluid, the ventricles a little more, and more upon the left side than upon the right. The piece of drainage-tube lay upon the floor of the fourth ventricle in such a manner as to leave a track along the under surface of the left lobe of the cerebellum. The arachnoid at the base of the brain was thickened, but it did not present any nodules, and it was distended into a sac containing clear cerebro-spinal fluid.

The effect of reducing the intracranial pressure in this case was to cause so marked and lasting a fall in the patient's temperature that latterly upon each occasion when it began to rise we looked for some obstruction to the free outflow of cerebro-spinal fluid, feeling certain that as soon as the stoppage was removed the temperature would fall again.

Case 2. A girl, *aet.* 5 years, was admitted into the Hospital 25th February, 1896. She had been under the care of my colleagues at different times for caries of the dorsal spine and for a psoas abscess. She was now admitted with a slight Bell's palsy, and with all the symptoms of tuberculous meningitis.

On February 27th I trephined the skull over the left lobe of the cerebellum, and as no fluid was obtained by puncturing the dura mater I

incised it. The cerebellum immediately protruded, but by the exercise of a little ingenuity I was able to pass a horsehair drain into the subarachnoid space. On the day following the operation the ward note says, "The patient is still semi-comatose, but she is not so fretful, and there are no more signs of facial paralysis." It says again on the next day, February 29th, "The patient is less comatose than before the operation, as she looks about and appears to notice things. The eyes show rather more signs of neuritis when examined with an ophthalmoscope." The temperature (Fig. 2.) fell

'Annie Scott - et: 5.

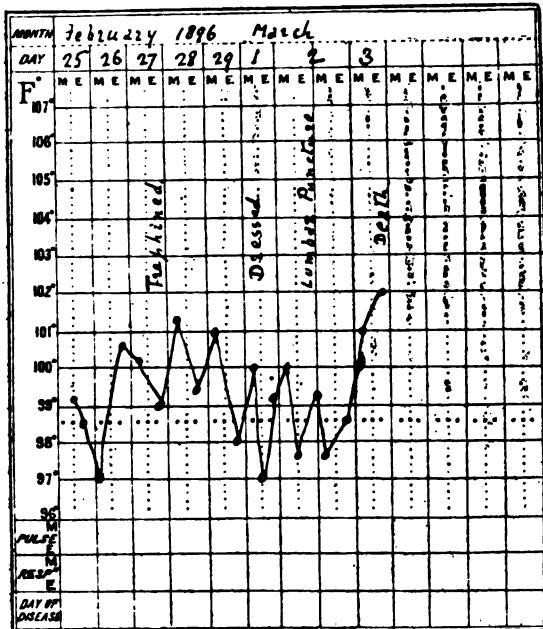


Fig. 2. Chart showing the fall of temperature following immediately upon lumbar puncture of the subarachnoid space.

steadily, and on March 1st the wound was dressed. There had been a considerable discharge of cerebro-spinal fluid, which had been absorbed by the extra thickness of the dressings which we had applied at the time of the operation. Some portions of the cerebellar substance were adherent to the dressings, so that had the child lived there would probably have been a hernia cerebri. The patient was worse on March 2nd, though there was still so free a serous discharge from the wound that the dressings, were soaked in it and they had to be removed. I therefore dressed the wound, which

looked perfectly healthy, but as the intracranial tension was still very considerable I punctured the subarachnoid space in the lumbar region as I have already told you (p. 53). The result of the puncture was to withdraw 200 minims of clear cerebro-spinal fluid. The temperature at 2 o'clock before the puncture was 99.2° F., at half past two, directly after the puncture, it was 97.6° F. (Fig. 2), so that the withdrawal of the fluid was coincident with a fall of a degree and three fifths in the temperature of the body. The pulse underwent no change. The patient's condition was not improved, the temperature soon began to ascend again, and the child died on the following day.

These two cases appear to show that a portion of the increase in temperature in cases of meningitis is due to intracranial pressure, and it is possible that a great deal may be learnt from a careful consideration of the temperature chart in these cases, though at first sight the oscillations appear to be perfectly irregular. The chief and permanent cause of the rise of the temperature in meningitis is, perhaps, more pathological than physiological, for it is probably caused by the deleterious effects of the toxins produced by the growth of the micro-organisms ; yet this could not be the cause in the first case I have related to you, for the cerebro-spinal fluid was sterile. The maximum temperature, especially in cases of tuberculous meningitis, immediately precedes death, and this points to the fact that the fatal ending is due rather to the action of the toxins, and to the impairment of the cerebral circulation, than to the paralysing effects of intracranial pressure. The removal of the cerebro-spinal fluid may act beneficially, especially in cases of infective meningitis which are not tuberculous, partly by relieving the pressure, partly by removing the toxins, and partly, perhaps, by so altering the chemical and physical properties of the fluid that the micro-organisms are no longer able to flourish. It is useless to trephine in cases of tuberculous inflammation, for the tissue changes are so great that even could the important cerebral conditions be removed there still remain the active foci of inflammation in other parts of the body.

The treatment in such cases can only be palliative. Opium, or morphia with atropine, may be given in full doses. They are well borne, and are, I think, more reliable than chlorides and the bromides, but Dr. Barr has recently

advocated the use of atropine alone, with the application of a blister to the scalp, or to the nape of the neck. Cold baths from 68° to 78° F. will sometimes be found to be of greater service than the administration of antipyretic drugs, for they lower the temperature, and in theory, at any rate, favour the elimination of the toxic products by increasing the secretion of the urine. Iced water may be sipped to allay the vomiting, and small doses of calomel often repeated will be found most serviceable in overcoming the constipation. These, however, are medical details, and they therefore come from me rather in the character of *obiter dicta*.

CLINICAL NOTES.

WITH DR. LUFF IN THE WARDS OF ST. MARY'S HOSPITAL.

Leucocythaemia.

THIS woman, whose age is about 45, is the subject of leucocythaemia, as you will have become aware from the examination of the excellent microscopical specimen of her blood. The disease may occur at any period of life, but is commonest between the ages of 30 and 40, rather more than one third of the cases being within this range. Another interesting fact is that it is more frequently met with in males, the sexual preponderance being as 2 to 1. The special age in male patients is from 30 to 40, one third being in these 10 years of life, whereas in females it is between the ages of 40 and 50; moreover, in females the state is associated with the climacteric period, because after the cessation of menstruation leucocythaemia practically does not occur in women. Dr. Gowers, I believe, managed to find one case of leucocythaemia in a female of 60 years of age, but its occurrence at that time of life is quite exceptional. Heredity, apparently, has no bearing whatever upon the disease. As to the actual exciting cause we really know nothing, except that a fair proportion of the cases are connected with malarial or intermittent fever; yet the majority of cases of leucocythaemia have not come into contact with the malarial organism, having lived

in districts where ague is not common, so that in most cases it is difficult to trace a cause of the disease. In 30 per cent. of the cases there is a history of preceding malarial or ordinary fever. When leucocythaemia does occur subsequently to malarial fever, it is a very late sequela. If, in malarial cases, the leucocythaemia is seen early enough, there is every prospect of affording relief by treatment, but, unfortunately, the majority of the patients are not seen until it is too late to afford them any benefit.

The symptoms are simple. There is pain in the left side from enlargement of the spleen, and with that is generally associated gastric disturbances, patients complaining of indigestion, pain about the stomach after taking food, and flatulence. The other symptoms are those of anaemia—since the disease is associated with profound anaemia—viz. breathlessness, especially upon exertion, and signs of cardiac failure.

The diagnosis is a simple matter, viz. enlargement of the spleen plus the leucocythaemia—the detection of the abundant leucocytes in the blood. Leucocythaemia has been mistaken for Hodgkin's disease, but examination of the blood should always settle that. Of course, you may meet with cases of anaemia and chlorosis in which there is a large increase in the white corpuscles, and therefore it is necessary to know the line of demarcation between leucocythaemia and these diseases. It is generally agreed that one or more white corpuscles to every twenty red constitutes leucocythaemia, when associated with enlargement of the spleen.

Then with regard to the pathology of the disease, what is the cause of this leucocytosis? It is that the antecedents of the corpuscles, instead of becoming red become white; that is, leucocytes are formed in leucocythaemia from the material which, in the normal state, would go to make red corpuscles. Leucocythaemia is sometimes associated with enlargement of the lymphatic glands, and it is also apt to be associated with disease of the medulla of bones. In other words, all the organs or parts concerned in the manufacture of blood-corpuscles are affected. Now, if from disorder of the function of the spleen, or of the marrow of the long bones, the haemato blasts are not converted into red corpuscles, they become leucocytes, and are partly discharged into the

blood, thus producing leucocythaemia, while those which remain in the organ cause it to swell. The size of the spleen in this disease is not merely due to this retention of these leucocytes, but also to an actual increase in the splenic pulp. That is practically all that is known about the pathology of leucocythaemia.

Next, as to prognosis. In the vast majority of cases patients do not seek medical advice until the disease has progressed considerably. After recognition it runs its course in from a few months to a few years, the average time being two years. If patients come for treatment early enough there is no doubt that, although the disease can never be cured, it can be very considerably ameliorated; that is, if they come at a time when the spleen can only just be felt below the ribs, and if the white corpuscles are not in a much greater proportion than 1 in 18 or 1 in 15.

As to treatment in these early cases, the first desideratum is to bring about contraction of the spleen; and this can be accomplished by the use of icebags, by massage, and by galvanism of the spleen; this treatment also diminishes pain and discomfort. A spleen of the size and consistence of this patient's will never contract. Quinine and arsenic are the two principal drugs used, but it is doubtful whether arsenic is of much benefit. Quinine is of use where the leucocythaemia is of malarial origin, and in those cases it should be pushed. There is another drug which is useful in an early stage, on account of its causing contraction of the involuntary muscular fibres of the spleen, viz. ergotine, or the liquid extract of ergot. In an early stage of leucocythaemia, in addition to employing massage and electricity, give quinine in 5 or 6 gr. doses with liquid extract of ergot. The diet should be good, and the patient kept at rest so as not to disturb the heart, which is itself in a bad condition in this disease. Of course the prospect of cure in these cases should never be held out.

A pertinent question is : Is this a case in which you would advise removal of the spleen? The answer is, no. It has been tried in such cases, but the tendency to haemorrhage is so great that such an operation is fraught with immediate danger. Such patients resemble the subjects of haemophilia in the tendency to excessive bleeding from very slight causes. Any operative interference, even

such a small matter as drawing a tooth, should be approached with very great caution. One of the commonest lethal terminations of this disease is due to haemorrhages, such as epistaxis, bleeding from the gums, haematemesis, uterine, and even vesical haemorrhages; and, in addition, there may be subcutaneous, submucous, and subserous haemorrhages.

I would add one other note of warning,—never squeeze or tie a finger to get blood out in these cases, because by such a course you are apt to disturb the proportion of liquor sanguinis and corpuscles.

Jaundice due to Impacted Gall-stone.

This woman was admitted eight days ago, and that she has jaundice is quite obvious. She appears, during the last few months, to have had two or three distinct attacks of pain in the epigastric and right hypochondriac regions, accompanied by severe vomiting, the vomit being dark green in colour.

Twelve months ago, the patient had jaundice, but felt no pain or sickness; she, however, felt very weak and low-spirited. She has lost a considerable amount of flesh lately. In addition to the extreme yellowness, which is most marked on the abdomen, there is a purpuric rash. At present she is free from pain and sickness, but complains of a nasty taste in the mouth. There is tenderness to pressure in the right hypochondriac region, and the liver can be felt down to the umbilicus. The spleen is not enlarged. Urine 1020, reaction acid, no albumen or sugar, but bile is present. The patient tells us that the pain seemed to shoot straight down to the pubes. There is no elevation of temperature, therefore we may exclude the question of pus formation. The history of the case leaves little doubt that the trouble is caused by an impacted gall-stone, probably of considerable size. I should think that since her previous attack of jaundice she has had one or more gallstones in the bladder, and that one has now slipped into the duct and blocked it, because the motions are free from colour.

This is certainly a case where we ought to have a consultation with the surgeon, to see whether operative interference is desirable. I think it is to her interest to have at least an exploratory incision.

Hypertrophic Fibrosis of the Liver.

This man is *aet. 40*, and is the subject of hypertrophic fibrosis of the liver. There is resonance over the umbilical and hypogastric regions. The abdomen in this case was tapped five days ago, and ten pints of fluid withdrawn. The tapping now requires to be repeated. Probably ten days or a fortnight will elapse before a third tapping is called for, and after that it may only require doing once more. Then the patient may go on comfortably for a long period. In these cases where there is a very large amount of fluid in the peritoneal cavity, and the blood, lymphatic, and other vessels have been extremely stretched, the first tapping causes a great relaxation of the vessels, and in consequence, a very rapid re-accumulation of fluid occurs.

The condition of this man is due to hypertrophic fibrosis of the liver, not to atrophic cirrhosis. Most cases of liver affection from alcoholism are hypertrophic fibrosis; the instances of atrophic cirrhosis of the organ are comparatively rare. The latter condition is called hob-nailed or gin-drinkers' liver, and is due to the taking of large quantities of neat spirit. When diluted alcohol in persistently large doses is taken, whether in the form of beer or spirituous liquor, in the great majority of cases the liver becomes hypertrophied. Cirrhosis is an unfortunate term for the affection, because one is apt to associate it with contraction of an organ.

Hypertrophic fibrosis of the liver is one of the painful diseases of that organ when it is pressed upon, though the patient does not suffer pain in the absence of pressure from without. Enlargements of the liver should, for convenience of diagnosis, be divided into painful and painless, the former comprising those cases in which a sudden push on the abdomen in that region causes the patient to wince. The painless enlargements of the liver are the following, in the order in which they are likely to be clinically encountered with the greatest frequency, a point you will do well to bear in mind in the enumeration of any diseases:—

(1) Fatty liver, due to the accumulation of fat in the liver. Excessive beer-drinkers sometimes have fatty liver instead of fibrosis; we do not know why, but perhaps fibrosis is present oftenest in excessive beer-drinkers, fatty liver being more

confined to those whose indulgence has not been so free. This patient took both beer and diluted spirits, but more beer. Persons suffering from fatty livers are usually those who, while eating heavily, take very little exercise; hence it is found among the leisured classes, whose only exercise, in many cases, consists of driving.

(2) The next commonest painless enlargement of the liver is albuminoid or lardaceous disease. That is particularly seen in children suffering from some wasting disease, such as suppurative bone disease, psoas abscess, or a chronic suppurative malady.

(3) The third kind of painless enlargement is much less common than the preceding two—hydatid enlargement of the liver. Such patients come sometimes with an enormous liver, but quite unaware of any disease, though it may have been going on for months. If hydatid liver be associated with pain, you may safely conclude that suppuration is in progress.

(4) A very rare painless enlargement of the liver is simple hypertrophy. It would be impossible to diagnose this by itself during life. The patient with leucocytæmia, whom we have just seen, has a large liver, and that is probably an instance of simple hypertrophy—there is no increase of fibrous tissue between the nodules.

Painful enlargements may be due to congestion of the liver of some sort, but the enlargement would then be secondary to the congestion. Congestion of the liver may be of three kinds. If the congestion results from taking highly-spiced food or alcohol, or from taking any irritant in the food, it is called active congestion, the irritant being conveyed from the stomach and intestines in the portal blood. Secondly, congestion may be due to fulness of the vessels of the liver from the back-working of the blood, either from obstructive lung disease or obstructive heart disease. It occurs especially in regurgitant heart affections, and in cases in which there is failure of compensation in the heart. That is termed mechanical congestion. Again, the liver is supplied by portal blood, as well as ordinary blood, and when the flow of portal blood is stagnant we get the third, passive congestion. This last form is met with in chronic constipation, and it is a point to be borne in mind in the treatment of chronic constipation, that not only must the tone of the intestines be

restored, but the torpidity of the liver must be removed; in such cases massage is extremely useful.

From congestion we naturally pass to inflammation. Enlargement of the liver may be due to some form of inflammation, either diffuse hepatitis, or perihepatitis. Enlargement of the liver is never very great from perihepatitis. Inflammation may be due to a severe blow, or to some poison circulating in the blood, or to specific disease. Perihepatitis is fairly common.

The next painful enlargement of the liver is due to pus. There are only two forms of liver abscess: pyæmic abscesses, which are always multiple, and tropical, which is always single. This latter, of course, can only result from tropical disease, and considerable swelling and fluctuation are noticed. Tropical abscesses vary in size from a small orange to a cocoa-nut. The pyæmic abscesses are of course due to pyæmia, and are frequently started by dysentery, ulceration of the large intestines being the source of the pus.

Apart from abscess of the liver, there may be primary or secondary deposits in the organ, *i.e.* carcinoma or sarcoma; or a large amount of fibrous tissue may be deposited there, as in the patient before us. Hypertrophic fibrosis of the liver is especially apt to be due to active congestion, particularly that set up by alcohol.

Pyelitis.

This man is 52 years of age, and complains of indefinite abdominal pains, especially in the region of the bladder. His occupation is that of night watchman. The history is that about two months ago he felt quite well, but soon after he began to suffer intermittent sharp pains in the abdomen, and noticed that his urine was thick and rather light coloured. These symptoms, accompanied by a feeling of general weakness, continued until last Friday afternoon, when he was seized with a sudden fit of shivering, which lasted 45 minutes. Subsequently he perspired very freely, but was better on the following day. When seized with the rigor a very severe pain shot up both arms, as high as the clavicle. His chief complaint now is the presence of pain in the hypogastric region, but he has a burning pain in the urethra on micturition. He also has a cough. There is no nausea or vomiting; the abdomen is somewhat distended, and pain is

indicated between the pubes and the umbilicus. The tongue is large and tremulous, coated on the dorsum, and the abdomen moves excessively during respiration. There are no dilated veins. Resonance is good all over. The liver is felt below the costal margin; dulness extends as high as the fifth rib. There is no tenderness over the gall-bladder, nor over either kidney. Urine 1010, light straw colour, albumen one-tenth; no casts; no blood; reaction alkaline; not offensive; deposit of phosphates. On examination of the chest, the vocal fremitus is equal on both sides, but there is hyper-resonance, probably due to a little bronchial emphysema. The alkalinity of the urine must be due to either a fixed alkali, or to carbonate of ammonia. If the latter, it is due to the conversion of urea into carbonate of ammonia; I think that is the case here.

He may be suffering from cystitis, Bright's disease—subacute nephritis, or pyelitis. If from Bright's disease, we should probably have found some blood in the urine. The patient has never noticed his urine to be either smoky or red. Pyelitis—inflammation of the pelvis of the kidney—is accompanied by rigors, and by a small quantity of albumen in the urine; moreover, the urine is alkaline in reaction. All those factors are present in this case. In pyelitis, the deposit in the urine nearly always contains pear-shaped and tailed cells.

Therefore I hope you will make a very careful microscopical examination of the urine, keeping a sharp look-out for casts, and for bladder epithelium. A very good plan, apart from examining the deposit just as it is, is to take some of the urine and add enough acetic acid to dissolve the phosphatic deposit; then allow the urine to stand, and examine the deposit. That method will bring out the casts very much better than the ordinary plan.

In the last issue of the CLINICAL JOURNAL, No. 3, Vol. VIII, p. 48, line 34, 3^{xv} should have read 3^v. The passage corrected runs: "In slight cases [of haemoptysis] Dr. Wethered recommends—

Tinct. Hamamelis 3^{xv},

Acid. Sulphuric. dil. 3^v,

Aq. 3j.

Ter die sumend."

A THURSDAY CONSULTATION AT ST. BARTHOLOMEW'S HOSPITAL.

(?) Loose Body in Knee-Joint.*

Mr. BOWLBY : This patient is a young woman of 23, admitted on account of the condition of her knee. Her knee is painful when she walks. There is no apparent disease, and she has suffered four years from pain on movement, and the knee giving way after walking a short distance. There is nothing to be seen on inspection, but as soon as you move the knee, you can feel and hear a slipping on flexion. There is so much pain that she has applied here in order to get relief, and the question is, what can be done by operation or by instruments to prevent this movement and to remedy the pain of which she complains? You can hear the sort of dull thud on the patient flexing the knee.

Mr. BUTLIN : This condition has lasted for four years; it came on quite suddenly, and there are no other creaking joints. I have seen the temporomaxillary joint affected much in the same way.

Mr. BOWLBY : The click is caused apparently on the outer side of the joint. The question is, is it a movement of the tendon or ligament, or is it the external semi-lunar cartilage? Mr. Marsh is inclined to attribute it to the external semilunar cartilage. The point is now, what are we to do to the woman to remedy the condition? The history of the case is that the patient originally in March, 1892, got out of bed and found herself unable to straighten her right leg. This lasted for a week, when she found she could straighten the leg, but every time she did so something gave way, with the jerk and the noise we have heard. On straightening the leg she had a good deal of pain during the first few days, but afterwards that pain was not severe, except after she had walked some distance. I may say, first of all, that Mr. Marsh's opinion is that it is probably a loose external semilunar cartilage, and he is inclined to advise an operation.

Mr. T. SMITH : I feel sure from what I hear from the house surgeon in charge of the case that there

is a loose body which has been seen and felt. With the assurance that there is a loose body in the joint from the house surgeon, in that case we are certain that an operation should be performed to relieve the condition.

Mr. BUTLIN : The symptoms are not classical symptoms of detachment of the external semi-lunar cartilage, or of a loose body in the joint. There is a kind of a jump as the knee is flexed. It seems to me that it would be best to open the joint and see what the condition of things is. I think myself that it is a condition which could be cured by an operation.

Mr. BOWLBY : As far as the symptoms in this particular case are concerned, they are different to those usually met with in the case of a loose body in the knee-joint, for with the exception of the single first attack, this patient has never been laid up with synovitis. The usual history is that from time to time the patient has a very sudden attack of very severe pain, with locking of the joint and effusion into the joint; that has not been present in this case, so under these circumstances I feel diffident about making any exact diagnosis; it may be that the tendon of the biceps slips in the movement of flexion of the joint, but I feel quite certain that an operation should be done to remedy this condition.

Congenital Malformation of both Hands.

Mr. BOWLBY : This is a patient of Mr. Willett's, a man æt. 29. He has come in for a deformity of his hands, which you can see for yourselves; it is stated that he was born with his hands in this condition, except that the fingers were tightly flexed into his hand. Both hands are almost similarly deformed, the fingers are flexed at right angles to the metacarpals and abducted; as a rule the wrists are kept hyper-extended, and he is unable to flex the hand on the forearm. I think an operation might be performed to remove the prominent heads of some of the metacarpal bones, but if we succeed in enabling the patient to extend the fingers more I think it would be something. The idea is to operate on one or two of the heads of the metacarpal bones first, and see the results before continuing the operation.

Mr. T. SMITH : I think that the only way to cure the deformity is to take off the heads of the metacarpal bones. The patient tells me he can write

* 5, STRATFORD PLACE.

DEAR SIR,—It may be interesting to you to know that the knee-joint trouble proved to be a loose external semi-lunar cartilage.—Yours truly,

T. SMITH.

To the Editor, *Clinical Journal*.

pretty well, and that the great drawback is the appearance of the hands; but it is a question whether an operation of this kind would not injure his writing, and this point is worth considering as his occupation is that of a clerk. To make the hand look better you would have to remove all the heads of the metacarpal bones, and one hand should be operated on first of all.

Mr. BOWLBY: We should begin with one hand first.

Mr. T. SMITH: I think it would be better to begin with the hand he does not use for writing.

Mr. WALSHAM: It is proposed to take off the heads of the metacarpals, much in the same way as you treat a hammer-toe. The patient himself said that it was the right hand he wished to be operated on, because he can keep the other hand in his pocket out of sight. I do not think the operation would interfere with his writing, and I should certainly recommend that the operation should be done.

Mr. LOCKWOOD: I think it is a congenital condition; there is also considerable webbing of the fingers, moreover; there is a specimen in the museum of a foetus which was born with similar hands. I think it would be a better way to begin on his left hand first, and if that is successful to proceed further; as he can write so well, I should only operate first on his left hand.

Epithelioma of the Jaw.

Mr. BOWLBY: This patient has been recently under the care of Mr. Adams, and he asked me to see her. She has a swelling in the mouth, on the right side; you can see it on the right lower jaw commencing immediately to the outer side of the incisor teeth. There is a soft spongy swelling extending to the place where the molar teeth would be. This swelling is quite soft and spongy to the touch, and presents no induration; there is no suppuration in progress, and there are no teeth at the site of the swelling; the jaw-bone beneath the swelling is distinctly diseased, part of it is already gone, there is no thickening and no enlargement to be felt. The appearances of the thing suggest *actinomycosis*, but I may say at once that I have removed a portion of the swelling, and under the microscope it is a typical epithelioma. The curious part about the case is that clinically it does not present the characteristics of epithelioma—it is a quite soft spongy swelling, there are no lumps or hard margins, and no enlargement of the glands. It is unlike an epithelioma in most respects; under the microscope there is certainly epitheliomatous growth, and considering that it has been three

months growing, and has extended rapidly, it probably is clinically an epithelioma. So far as the cause is concerned, there is one suggestion to be made in connection with this tumour: there is a tumour, a multilocular cystic epithelioma of the jaw, which, occurring in the bone, does not run the course of an ordinary epithelioma. Clinically this tumour cannot be distinguished from epithelioma, it begins probably from some foetal remains; these run a different course to the ordinary epithelioma, and clinically present a soft spongy swelling. I have already mentioned that, as far as microscopical examinations go, this patient's tumour is an ordinary epithelioma, originating in the surface and extending into the bone. The indication is, that if the growth springs from inside the jaw it would not be a malignant growth.

The point is, what shall we advise this patient to have done, and if an operation is determined on, to what extent shall the bone be removed. I may say that I took the view that the growth is one probably extending into the bone, but not springing from inside the bone; it has grown more rapidly than the multilocular cysts of which I spoke of, and further, the patient is somewhat advanced in age. I think there can be no doubt that this is malignant, and as to any operation I am inclined not to remove the tumour through the mouth. I think an incision along the lower border of the jaw will be necessary, but I do not propose to cut away the whole depth of the jaw-bone. Of course I shall be guided from what I see at the operation, and I think she should be operated on early. From the mouth it cannot be got out so satisfactorily as it would be in the way I suggest.

Mr. T. SMITH: I think it is an epithelioma of the lower jaw, and not a cystic growth. Cystic disease does not grow so fast as this has, and though I understand that the cystic growths are called cancerous, yet I have in my own practice a patient who, certainly, for the last thirty years has been operated upon several times. In this case the jaw should be laid bare to the symphysis, and as one or more teeth are loose, I should cut the jaw away clean from the symphysis. It seems to me a rapidly increasing growth.

Mr. WALSHAM: I think it is epitheliomatous; and as the growth is spreading, I should do the operation very widely, but if possible leave some portion of the lower margin of the jaw.

Mr. LOCKWOOD: It must be an epithelioma, relying on the histological examination, and I should myself attempt to remove it from the mouth, particularly if you are not intending to remove the whole jaw.

Mr. BOWLBY: I should say we are all inclined to an operation, but under the anaesthetic we shall be able to decide more particularly as to the extent of the operation; one thing is certain, that the growth must be very freely removed.

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 27, 1896.

CLINICAL LECTURE ON SOME CAUSES OF HÆMATURIA.

Delivered at St. Thomas's Hospital, Westminister, S.W.,
ON

Wednesday, November 11th, 1895,

By SIR WILLIAM MAC CORMAC,
M.A., D.Sc., F.R.C.S.,

Consulting Surgeon to, and Emeritus Lecturer on Clinical
Surgery at St. Thomas's Hospital.

GENTLEMEN,—The subject of lecture to-day is the interesting and important one of Hæmaturia. Hæmaturia may result from different causes ; and I have here a number of cases, which I will read to you in abstract, illustrating the different sources from which blood in the urine may come. For instance, in this case of carcinoma of the bladder there is a history of bloody urine for eighteen months, the blood being always bright, sometimes in large clots, accompanied by a great deal of pain and frequent micturition. There is also pus in the urine as well as blood, and it is alkaline. The patient, a man æt. 59, was sounded for stone without result ; a supra-pubic exploratory cystotomy was then performed, and subsequently a permanent drain was introduced ; no radical operation could be performed. The disease proved to be a large cauliflower growth on the posterior upper part of the bladder, too extensive to be removed.

The next case was that of a man, æt. 45, with a villous papilloma of the bladder. Recurring attacks of hæmaturia had continued for two years. These came on suddenly without pain, the amount of blood being often large, while for considerable intervals the urine was quite normal. The blood was always bright in colour, passed towards the end of micturition, and trickled away afterwards, — points of some importance.

The next is a case of vesical calculus in a man æt. 72. The symptoms had existed for seven months ; the micturition was frequent, the hæmorrhage intermittent, frequently excited by any un-

usual exertion. The blood was bright in colour, mixed with the urine, sometimes abundant and clotted. The stone was discovered with the sound.

The next case is one of tuberculous disease of the prostate. The patient, a man æt. 26, suffered from increasing frequency of micturition for a year, and during the last six months of the year from hæmaturia. The haemorrhage was never severe, often so slight as only to be detected by the microscope—a point of importance in connection with tuberculous disease. It was also intermittent. The urine was loaded with urates and pus. Severe pain was felt in the region of the prostate—which was large and tender,—and the perineum, and a nodule was felt in the epididymis, probably tuberculous. No treatment could be adopted in this case of a radical kind.

The next instance I wish to mention is of hæmaturia due to tuberculous cystitis. In this case there was chronic inflammation of the bladder in a man æt. 31. The pain, frequent micturition, and hæmaturia had lasted for five months. The bleeding was slight in amount and very variable in its incidence. The urine contained pus.

The next case is that of a man, æt. 52, who had stricture of the urethra, accompanied by frequent hæmaturia. The blood was passed at the beginning of the act of micturition, which was of great frequency.

In addition, vesical haemorrhage often takes place after emptying the bladder in cases of acute retention ; bleeding from the urethra may be due to rupture of the tube from accidental causes, such as blows on the perineum, fracture of the pelvis, or the impaction of calculus in the urethra ; and bleeding occurs from the irregular use of instruments, or from a false passage made by a catheter.

Then we come to the hæmaturia due to sources further removed. The first case is that of a man with a papillomatous growth in the kidney. He had a history of calculi six years before, with renal colic, and for the last eight months hæmaturia. The hæmaturia was excessive ; it resisted all medicinal influences, caused general anaemia, and the blood, as you might expect, was always uniformly

mixed with the urine. Supra-pubic cystotomy was done in this case, and the blood was then seen to leak into the bladder from the left ureter. An exploration of the left kidney was then made, some calculi extracted, and the growth scraped away, which proved on examination to be a papilloma. The patient was cured.

In a case of renal calculus, the man, æt. 66, experienced lumbar pain for a month before coming under observation, and a swelling in this region was noticed a few days afterwards. The blood was never large in quantity. The urine was acid, and mixed with pus. On examination a tense, semi-fluctuating elastic swelling was found in the left lumbar region, extending to the umbilicus. On aspiration this yielded a bloody fluid, which contained no urea. Subsequently lumbar nephrotomy was performed, and a large multilocular cyst with an impacted calculus found. The patient was ultimately discharged with the sinus remaining open in the lumbar region.

The next case was that of a female, æt. 33, in whom a tumour was discovered in the left loin. There was blood and pus in the urine. There had been great pain in the lumbar region for ten months, renal colic, and intermittent haemorrhage varying in amount, the blood being always uniformly mixed with the urine, which contained pus. An exploration of the kidney was made, and offensive blood, urine, pus, and calculi were evacuated. The cavity, which was found to consist of loculi, large in size, with retained pus in them, was drained. This patient was discharged with a sinus open in the loin.

The next case, a man æt. 23, is one of tuberculous kidney, in which a fluctuating swelling was found in the left loin. The swelling extended from the ribs to the iliac crest. There was a history of renal calculi six weeks before, and intermittent passage of blood, small in amount, and sometimes only to be detected by the microscope. An incision was made in the lumbar region, pus evacuated, and an abscess found extending up beneath the diaphragm. The pleura was accidentally opened, became infected, and the patient died, gradually exhausted. Suppuration round the kidney and tuberculous foci in the pelvis were discovered post mortem. The left pleural cavity contained a loculated collection of pus above and an empyema below. The prostate was also found to be infected with tubercle.

These cases serve to illustrate some of the causes which give rise to hæmaturia or bloody urine; and you can deduce from these histories how important a symptom it is, and how many different conditions of disease it has relation to.

The blood, from wheresoever it may come, mingled either intimately or less intimately with the urine, finds exit in all cases at the urinary meatus. It may come from any part of the genito-urinary tract, either from the secreting structure of the kidney or its pelvis, from the ureteral tract, from the bladder or prostate, or finally from the urethra. You may take it, as a rule, that the more abundant the blood is in quantity and the nearer it is to its natural colour, the nearer probably is the source of the bleeding to the external orifice.

Speaking quite generally with regard to the causes of hæmaturia, it may be the result of local damage or irritation in some part of the urinary tract; caused by injury, by the presence of calculus, by congestion or inflammation; it may depend upon a simple or upon a malignant growth, or upon a general disease; it follows as a complication in some of the eruptive and continued fevers; it occurs in scarlatina and small-pox; it is found in purpura; it is also a complication, but very rarely, of scurvy; and it may occur as the result of some change in the circulating medium itself. In hæmophilia—in patients we call bleeders—haemorrhage from the genito-urinary canal does sometimes occur, but it is rare. There is a curious disease called hæmoglobinuria, in which the urine contains blood elements, but few if any corpuscles; the colouring matter is found in great abundance, and apparently leaks into the urine from the kidney tissues. The disease, more of a medical than a surgical nature, and due to some blood change, is called paroxysmal hæmaturia. I may mention one case in a man æt. 37, who had hæmaturia for six days. It came on with sensations of cold and chill. The urine was of a deep claret colour, and no blood-corpuscles could be seen under the microscope. The urine contained the colouring matter of blood, but no corpuscles. The specific gravity was 1010, and it was quite acid in reaction. There was considerable pain felt in the loins.

In hæmaturia proper, the red corpuscles are the colouring material, and these mingled with the

urine give it either a smoky tinge, a red or chocolate colour, or even a dark porter-like hue. Further, we may have haematuria as the result of the administration of drugs, like cantharides or turpentine; from excessive drinking of such liquids as beer, which I have known to cause excessive haematuria; and then we have cases which depend upon injury of the kidney, either contusion or wound.

In cases where blood comes from the urethra, it is nearly always associated with the presence of a stone. In the bladder we may have bleeding arising from the presence of calculus or the ulceration caused by tubercle, from villous or carcinomatous growths, from the dilated vesical veins of old persons, or from the prostatic veins in enlargement of that organ. We have an additional cause of haematuria, generally vesical, produced by the ova of a form of trematode worm in the bladder wall. This parasite is found in the drinking water of some tropical countries. When introduced into the system, the ova of this trematode find their way to the bladder wall, and occasion irritation there which produces considerable loss of blood, and gives rise to so-called endemic haematuria.

The amount of blood passed has an important diagnostic significance. It is, as you may suppose, of widely different amount in different instances. There may not be enough to produce any change in the urinary secretion visible to the naked eye; it may produce a slight smokiness due to the presence of a small amount of blood in the urine, the water passed may be pinkish in colour, a deep cherry red, or almost black—like porter,—or loaded with clots. Or the fluid passed may be like pure blood, the admixture of urine being completely concealed by the amount of blood. Under such circumstances it will often coagulate in the chamber vessel into which it is passed, or, what is more serious, may coagulate in the bladder itself.

The passage of blood may be continuous or intermittent. Clots when present sometimes indicate the part of the urinary tract in which the bleeding arises. For instance, you occasionally find clots leech-like in form which have probably been formed in the deeper urethra, whilst cylindriform pencil-shaped clots indicate the bleeding to have originated, and the blood to have assumed that shape, in the anterior part of the

urethral tube. The irregular amorphous clots probably form in the bladder cavity.

When the blood issues independently of micturition, or is mingled with the first portion only of the stream and clear urine immediately follows, you may assume the source is probably urethral. In vesical haemorrhage the urine may at first be almost quite clear, followed by a gradually increasing amount of blood as the act of micturition proceeds. When uniformly mixed, the blood is usually derived from the kidneys. In all these cases we must search for local indications of the source of the bleeding, local pain, and tenderness either in the loin, the testicle, perineum, or supra-pubic region. We also enquire into the existence or non-existence of calculi. The frequency and character of the stream of urine passed is of importance. We may get evidence of renal trouble or calculus, or a history of injury. The age of the patient and his previous history must be investigated. We may discover renal tenderness or tumour, a distended bladder, an enlarged prostate, or calculi may be felt in the bladder. In cases of new growth we look for some shred of substance in the urine, which—examined microscopically—will disclose its nature.

A word may be said here about the most common tests for blood. The fact that you may not actually see it with the naked eye, is no proof that blood is not present in the urine. The guaiacum test is easily applied and quite satisfactory, provided the tincture of guaiacum used is freshly made. It may be employed in two ways: either by adding to a few drops of urine, in a test glass, a drop or two of the tincture, and then a little ozonic ether, shaking them all together, when after the subsidence of the watery portion of the mixture, the ozonic ether will float upon the top with a well-marked blue tint; or if you mix the urine and guaiacum tincture together in the tube, and pour the ozonic ether on the top, you will see at the junction of the ether with the heavier fluid below, a distinct blue line when blood is present. The microscope is valuable as a means of detecting blood in the urine, for the blood-corpuscles will then be seen,—often altered in shape, either shrunken or swollen, and even globular in form,—sometimes containing colouring matter, and often without. In haematinuria, you will find no corpuscles, the corpuscles are broken down, and

the blood colouring matter causes the appearance of the urine. Of course the ordinary tests for blood produce the characteristic reaction, but the microscopic test will not be available.

The spectrum test is not so readily applicable. The absorption bands of yellow and green in the spectrum present two lines between D and E, which when seen denote the presence of blood in the urine. The crystals of Teichmann may be formed by treating the traces of blood-stains on the dress and so forth with acetic acid.

Urethral Hæmorrhage.—If we now consider the causes of hæmorrhage in detail, it may be convenient, perhaps, to take the hæmorrhage that occurs from the urethra first. This is not, strictly speaking, hæmaturic, because it occurs independently of the act of micturition and stream of urine. A frequent cause of urethral bleeding is rupture of the tube. A boy or man may be kicked in the perineum, or fall astride a bar or joist in trying to balance himself. In the adult, crush of the pelvis is frequently associated with rupture of the urethra. The pubic and ischial rami are the weakest parts of the pelvic girdle, and consequently in the injuries commonly called buffer accidents, if the pelvis be broken at all, those parts are sure to give way, and from their proximity the urethra is often ruptured, and bleeding takes place from it.

In some unfortunate cases, by reason of the unskilled use of instruments, a tear occurs in the mucous membrane of the urethra either during the introduction of a catheter, or the attempt to force a passage in the case of stricture, or a lithotrite carelessly employed may cause a tear, and in these circumstances blood flows from the urethra. A urethral calculus may become impacted in the tube, commonly in boys, causing irritation, inflammation and ulceration, and a certain amount of hæmorrhage from the raw surface.

In cases of acute inflammation of the urethral tube, in gonorrhœa for example, bleeding from rupture of the congested capillaries of the mucous membrane may ensue.

With regard to the diagnosis of bleeding from the urethra, one may speak pretty certainly of its source, when it appears irrespective of the act of micturition, or if the blood comes with the first jet of urine and the subsequent fluid is clear.

It is seldom necessary to interfere actively with bleeding from the urethra. Cold water applica-

tions, or iced water, or cold by means of an ice-bag externally, have proved desirable where the bleeding is considerable; cold water injections may be also tried; and in cases where the bleeding has proved excessive and cannot be otherwise controlled, a full-sized catheter, large enough to occupy the whole calibre of the tube, may be introduced, when by means of compression externally, the bleeding within will be controlled.

Prostatic Hæmorrhage.—Prostatic hæmorrhage in connection with enlarged prostate occurs from the varicose veins present in old persons. Very often a considerable amount of blood trickles back into the bladder, escapes during micturition, and frequently forms clots. On examining the prostate, and taking into consideration the age of the patient and other symptoms present, a diagnosis will be arrived at. The treatment will be mainly that adopted for enlargement of the organ.

In cases of prostatic hæmorrhage, arising during either acute or chronic congestion of the gland, or associated with ulceration, the blood does not pass down the urethra, but regurgitates into the bladder, and becomes mixed with the urine, and so far as symptomatic hæmaturia is concerned, is similar to that originating from the bladder itself. An investigation of the condition of the prostate through the rectum, and in other ways, will determine the probable cause of the bleeding.

Vesical Hæmorrhage.—The causes of vesical hæmaturia are many in number. We have calculus,—some forms, as a mammillated oxalate of lime calculus, being more prone to occasion bleeding than others. New growths in the bladder, villous papilloma, villous carcinoma or epithelioma, and tuberculous ulceration, are conditions which will be associated with the presence of blood in the urine.

Ulceration of the bladder may be produced by the too long continued presence of the catheter, often introduced too far. This sometimes will give rise to bleeding.

Hæmaturia will be generally found in association with calculus, and is more frequent after the patient has made some great effort, or when he is exposed to jolting exercise, and the calculus is shaken about in the bladder in consequence. Again, too, the bleeding may seem to increase towards the termination of the act of micturition,

for the reason that the bladder wall grasps the rough calculus by the contraction incident to emptying the bladder. The mucous membrane and the rough surface of the stone come into contact, react upon one another, and give occasion to an increased blood flow.

New growths in the bladder causing hæmaturia may be of different kinds. There is a simple mucous polypus of the bladder which does not cause any great amount of bleeding ; papilloma of the bladder is a villous, usually multiple growth. The hypertrophied villi are invested with a layer of thin epithelium, and contain a projecting capillary loop which is easily torn by the contracting muscular wall of the bladder. And hence we find in these cases abundant bleeding, frequently aggravated at the termination of the act of micturition for the same reason as in calculus—the contraction of the bladder wall upon the growth. The bleeding in these cases is sometimes so free that the patient loses large quantities, and may become blanched and exsanguine. The bleeding is not continuous, it recurs at intervals, and not only is the urine mixed with blood, but absolutely pure blood trickles from the urethra after the act of micturition has been completed, stains the invalid's shirt, and may flow for a considerable time. Then the hæmorrhage may entirely cease for a time, to recur either with or without any apparent exciting cause. The diagnosis is not very easy. The disease occurs in young persons, generally males, as the cases I have mentioned indicate ; a certain amount of roughness or irregularity of the bladder surface may be felt with the sound or catheter. You will seldom be able to ascertain the presence of tumour, either by rectal or bimanual examination. The growths generally occupy the fundus, and are frequently met with in the neighbourhood of the ureteral orifices. Occasionally we are fortunate enough to discover a portion of the growth passed in the flow of urine, or detached by the use of instruments, when a microscopic examination will declare its quality. In other cases the frequent micturition, age of the patient, and the character of the blood-flow will assist you in determining the necessity for an exploratory examination. This usually consists in a supra-pubic cystotomy, when the growth may be removed by nipping-forceps properly constructed for the purpose. The operation is considerably

hampered by the abundant hæmorrhage which occurs at the time, and sometimes very difficult to control : best, perhaps, by sponge pressure in the interior of the bladder. The blood prevents the growth being clearly seen, or removed so thoroughly as it is necessary to do in order to prevent recurrence. When multiple, as is frequently the case, there is difficulty in removing what is vulgarly called the root of the disease, and this renders recurrence frequent.

Of cancerous growths in the bladder, the most common kind is squamous-celled carcinoma, which may form on any part of the interior surface, and is not, generally speaking, amenable to successful treatment. The bladder wall becomes speedily infiltrated by the ingrowing tendency of the tumour, which soon ulcerates and produces at intervals most abundant hæmorrhages. As the disease advances, the general signs of carcinomatous influence on the constitution appear.

Palliative treatment alone is possible. It is scarcely practicable to excise portions of the bladder wall, and yet preserve life. Suprapubic cystotomy or perineal drainage affords a certain amount of relief,—all, unfortunately, we can do.

Tubercle in the bladder is also sometimes a cause of hæmaturia ; but in this, as in other urinary tuberculous affections, the amount of bleeding is not great. It presents the characters of vesical hæmorrhage already mentioned, and the diseased condition of the bladder is usually associated with the presence of tubercle elsewhere. There is such a thing, I believe, as primary tubercle of the bladder, but I have never met with an instance of it ; it is much more frequently either an invasion of the organ by the malady descending from the kidneys, or still more frequently by the disease travelling upwards from the epididymis along the vas deferens, and thence to the prostate and bladder. When it does occur in a primary form, it is as a deposit of miliary tubercles, which fuse together and produce small shallow ulcerations on the surface of the bladder, which bleed from time to time. The disease, as I have said, is generally associated with tuberculous deposit in other parts, and there is then no treatment for this condition apart from general treatment directed to the constitutional control of the disease.

A few cases have been treated by supra-pubic

cystotomy and scraping out of the diseased portions, with subsequent drainage. Great relief has been obtained for a time of the very distressing symptoms, and in some there appeared good prospect of a permanent cure.

Mr. Battle has recently published an account of a very successful case of this kind in which there was reason to consider the tuberculous disease had affected the bladder primarily.

Another form of vesical hæmorrhage is associated with chronic and acute cystitis, although it is usually transient and of no great amount.

We have occasionally a considerable quantity of blood accumulating in the bladder, dependent upon a varicose condition of the submucous veins of the bladder wall. These have been called vesical haemorrhoids, are met with in old people, and often in association with rectal haemorrhoids. They are a not infrequent cause of hæmaturia in pregnant women. In such cases the blood is often abundant, usually dark in colour, giving the secretion the colour of porter, and clots may sometimes be found in it.

Hæmaturia is occasionally caused by injury to the bladder—and from the ulceration produced by the pressure of a catheter which has been allowed to remain too long pressing against the bladder wall. One of the symptoms of rupture of the bladder is bloody urine. These cases will have to be discriminated by the history and other symptoms associated with them. In rupture of the bladder, the patient is unable to micturate, but the water on being drawn off will be found to contain a more or less abundant admixture of blood.

The early diagnosis of rupture is exceedingly important, and may be ascertained best perhaps by injecting the bladder with a measured quantity of fluid, which goes in easily enough, but on withdrawal by means of the catheter—through which the injection is made—a lesser amount or perhaps a very small amount of this injected fluid will be returned. It has passed out of the bladder into the peritoneal cavity or into the extra-peritoneal cellular tissue. There are other means of ascertaining the presence of rupture which I need not now dwell upon; I would only say that for the most part there is neither shock nor collapse. In six cases of the kind I can recall, this feature was conspicuously absent.

As regards the diagnosis of vesical hæmorrhage, one may say that although the first portion of

the urine drawn off by the catheter may be clear or contain a lesser amount of blood, as the act of micturition proceeds, or as the water being drawn off by a catheter continues to flow, the quantity of blood will increase so that towards the end there will be a much larger amount, and in some cases the blood only appears with the last portion drawn off or passed. Blood being heavier than urine gravitates to the lowest part of the bladder, and will be the last to be ejected.

In discriminating the vesical from other forms of hæmorrhage, you find on washing out the bladder the fluid on return will continue to be bloody, whereas if it comes from other places the fluid will sooner or later return perfectly clear and free from blood. The blood may be so abundantly poured out as to fill the bladder entirely with clot. In some cases blood derived from the dilated varicose submucous veins accumulates in such abundance as to distend the bladder with a semi-solid blood-clot, obstructing the passage of urine and causing great distress to the patient. This condition is one of great urgency, and the best plan of dealing with it is probably to pass in a catheter as large as the urethra will admit, break up the blood clot and inject fluid, so as gradually to wash the clot away. In the female, vesical hæmorrhage is more manageable than in the male, and its cause and the method of dealing with it may often be arrived at after a rapid dilatation of the short urethra, which affords ready means of access to the female bladder.

In the treatment of this form of hæmorrhage, cold may be applied either to the supra-pubic region or the perineum, or within the rectum or the bladder itself, and by the internal administration of ergot you may control the hæmorrhage in some cases where it is abundant. Rest is a most important factor, and it may be necessary to adopt some method of cystotomy in order to secure it. In addition one naturally makes an investigation of the cause in order to find how far it is amenable to direct treatment.

Renal Hæmaturia.—Going deeper in the urinary tract, we come to cases in which the ureter and kidney are the source of the hæmorrhage, and this form of hæmorrhage often presents points of resemblance to vesical bleeding, the more so as the affections which produce bleeding in the one case are frequently the cause of it in the other. The

bladder, the ureter, or pelvis of the kidney, may be invaded by similar pathological conditions. Moulded clots are very characteristic of bleeding from the ureteral tract above the bladder, but they are rare; they present themselves in the form of elongated cylinders. I do not think you need often look for them with the expectation of finding them. In the ureter, injury and calculus are the most common causes of bleeding,—not that calculus will always be associated with haemorrhage, but it is perhaps the most common of the rare causes of haemorrhage in this situation, and as I have said already, we have inflammation extending from the bladder up the tube, or ulceration present from whatever cause, simple or malignant.

Renal haemorrhage is generally associated with the presence of calculus in the kidney. We may have bleeding in connection with either acute or chronic congestion, and in various forms of acute and chronic inflammation of the kidney. Although often absent, we may have it in a marked degree in many cases of malignant disease of the kidney, either carcinomatous or sarcomatous, and in a lesser degree in tuberculous pyelitis.

When the blood comes from the kidney it is always intimately mixed with urine, and there are no clots unless in the accidental complication of a ruptured vessel or a ruptured aneurism in the kidney, which is very rare. If the substance of the kidney be the source of the haemorrhage, we commonly find blood-casts in the secretion, and microscopically kidney-epithelium is almost always to be found. And in accordance with the amount of blood the secretion will be either smoky, red, or dark in colour.

The haemorrhages associated with malignant disease, which has reached the stage of ulceration, are often exceedingly severe. They occur at intervals, and may be very profuse. In renal calculus, also, the bleeding is often abundant. We find it associated with casts, and we may be able to determine its cause from the other symptoms of calculus present. The urine is acid. Severe or dull aching pain is experienced in the loin on one side, and referred down to the testicle and the base of the glans penis. There is often a great difficulty in discriminating this condition from tubercle affecting the kidney. In tubercle the bleeding is less frequent, is generally scanty, and associated with the presence of pus.

We have to discriminate what may be called forms of Surgical Hæmaturia from that which takes place in a disease more of medical interest, viz. paroxysmal hæmaturia. One may mention the chief features of this malady in order that you may be conversant with it. It is characterised by sudden and irregular onset in the shape of a severe rigor, followed by discharge from the kidneys of urine loaded with broken-down blood. The disease occurs generally in adult males, and is often attributable to chill or exposure. Uneasiness is complained of, and often severe aching in the loins. The testicles are retracted, the temperature usually lowered 2 or 3 degrees; after half an hour or so the patient finds that the water passed is dark-coloured, often like porter in hue. The urine is faintly acid, and deposits abundant dark grumous material containing much albumen with granules and hyaline casts. The temperature then rises, the symptoms abate, the urine gradually clears, and in a few hours the patient feels quite well again. In the intervals of the attack the patient seems to enjoy his ordinary health.

On examination by the microscope, one may find a few blood-corpuscles, associated with abundant dark-brownish granular material, due to their general disintegration. The attacks vary in frequency and severity, are sometimes periodic, in other cases irregular. They may cause no great impairment of health or render the patient very anaemic. The disease seems to be distinctly dependent on the influence of cold, and to have some affinity with ague and Raynaud's disease. Some regard it as a kidney affection, although it has been more accurately described as a disease of the blood, and the complete breaking-down of the corpuscular elements seems to favour the latter view. So far as treatment is concerned, there is little or none of an active nature to be adopted. Prophylaxis seems to be the most important concern.

In all inflammatory affections of the kidney, in many febrile diseases, and as the result of irritant poison, there may be renal congestion and bleeding. You may have congestion and bleeding in a passive form occurring in cases of pulmonary and cardiac obstruction, usually of small amount and quite of secondary importance to the disease by which it is occasioned. In the early congestion of Bright's disease, hæmaturia is a frequent symptom. You also have it in the cirrhotic stage of the disease.

Referring again to the symptoms of hæmaturia associated with tubercle in the kidney, this disease either originates in the kidney in a primary form or occurs as part of a general tuberculosis. We have occasional haemorrhages in association with it, but these are quite uncertain in their onset, usually scanty in degree, and are often altogether absent.

Primary tuberculous disease in the kidney consists of grey granulations, which coalesce and subsequently caseate. When the kidney is affected in this way the disease frequently extends down the ureter to the bladder, or, on the other hand, it may be perhaps more frequently of an ascending nature, extending from the bladder upwards, having perhaps reached the bladder from a tuberculous epididymis. There is pain and tenderness in the loin, and very likely a tumour there. The bladder will be irritable and micturition painful. There will be pus in acid urine. Occasional haemorrhages take place, but, as I have already said, small in amount. The diagnosis is always difficult, and perhaps best arrived at by an examination of the secretion for the tubercle bacillus.

With regard to morbid growths in the kidney, of course these do not prove a source of haemorrhage unless ulcerated. The fibrous tumours sometimes met with in the organ do not cause haemorrhage, nor the sarcomata which occur in young children and infants. Carcinoma, either in the primary or secondary form, occurs in adult life, and is usually of a medullary character. It may prove a source of very extensive haemorrhage, which will produce an almost exsanguine condition of the patient. The disease is liable to be mistaken for renal calculus, and this is the more likely, because in the discharge you often find small calculous masses mixed with the blood, which have been passed down from the cancerous kidney.

Renal calculus is associated very constantly with hæmaturia. The calculus may arise from the amorphous urates, deposited in the renal tubules, or in a stellate mass of acicular crystals, and urate of soda may be found embedded in the kidney substance. Uric acid in solitary or clustered crystals may form in the tubules and constitute minute calculi. More rarely we have carbonate of lime deposits. In these cases the urine is generally acid in the earlier period of the history of the patient, and remains so for a long time; there may or may not be pus, and unless

the urine be ammoniacal, there will be no phosphates. The concretions when small in amount get washed away in the stream of urine, or remain to form the nuclei of renal calculi, which often reach a great size. The hæmaturia is variable in amount, from a slight smokiness to a very considerable quantity, and it is usually intermittent. In rough movements, such as riding, jolting in a vehicle, or even railway travelling, jumping, or any concussion, the amount of blood will be increased. This is rather important evidence of the presence of calculus; it is not conclusive, but it would induce you to look for other signs of renal calculus—pain in the lumbar region shooting downwards to the testis, which is often retracted, frequent desire to micturate, pain at the end of the penis, nausea and sickness. By the microscope blood-corpuscles will be detected, and crystals of the urinary deposit similar to that helping to form the calculus. We have frequently a history of colic, called renal colic although the pain is produced by the passage or lodgment for a time of a small calculus in the ureteral tract; while a calculus is passing through this it causes agonising pain, often associated with severe nausea, sickness, vomiting, cold sweats, and sometimes collapse. The pain commences quite suddenly, and terminates as abruptly. One is reminded of the candidate who was asked by an examiner about the condition of a patient suddenly seized with renal colic, and the symptoms of passage of a stone along the ureter. "All at once," said the examiner, the patient exclaims, "I feel as if I were in heaven;" what has happened?" "Oh," said the candidate, "he is dead." Complete relief is experienced as soon as the stone reaches the bladder. Renal haemorrhage may follow the evacuation of the bladder in cases of prolonged retention. The backward pressure to which the pelvis and calyces are thus subjected is suddenly removed, and this permits of an intense congestion with subsequent haemorrhage from the mucous membrane lining the pelvis and calyces. Some of the capillaries probably give way under the strain of the blood suddenly accumulating in them, consequent on the removal of the intra-renal pressure. No blood will be noticed on the first introduction of the catheter, but on each subsequent occasion the urine will be found to be bloody. The amount and duration of the haemorrhage varies in different

cases. It is a not infrequent accident after retention in old persons, and may in them be followed by some form of nephritis. In young persons, otherwise healthy, no serious consequence need be entailed, and after a day or two the blood will diminish and disappear.

In the more severe forms vesical drainage may be required, especially if the blood accumulates and forms clots in the bladder.

An interesting form of haemorrhage from the kidney or some part of the urinary tract is dependent upon a parasite frequently met with in Egypt, Abyssinia, the Cape, Natal, and various parts of Australia. This form of so-called endemic haematuria depends on the presence in the urinary tract, very commonly the bladder wall, of the ova of the *Distoma* or *Bilharzia hematobia*. This cause may be suspected if you meet with cases of haematuria in this country in persons coming from places where Bilharzia is common. It may be diagnosed by the presence of the ova in the shreds of mucus passed in the urine. The parasite is derived from drinking the water of the Nile or other rivers where the eggs or larval forms of this entozoon abound. The Bilharzia is a trematode worm, a species of fluke, one variety of which causes the rot in sheep, and other kinds when present in man very often occupy the biliary passages; but this particular form—the *Distoma hematobia*—is met with when in a mature shape as a worm 12 or 14 millimetres in length. The male has a flattened body, while the female is almost cylindrical. The entozoon may be found in large numbers in the inferior cava and its tributaries. The ova, often present in large numbers, are of a peculiar form with a spine attached at one end, which serves to fix it to the mucous membrane either of the bladder or intestine, and occasionally the ureter or pelvis of the kidney. When found in the intestines it gives rise to dysenteric symptoms, and in the urinary tract it causes haematuria and cystitis.

The life history of this parasite is not very accurately known. We do not know even how long it lives, and with regard to the treatment, removal from the source of the disease is the only one available. Care should be taken that persons living in the infested district should not drink water which has not been previously thoroughly boiled and filtered, so as to destroy the ova of the entozoon.

Then in some parts of the world, in the Indies, both East and West, there is a peculiar kind of nematode worm—the *Filaria sanguinis hominis*—which infests the blood, and occasionally produces bloody urine, probably of kidney origin. The embryo of this parasite possesses a long slender body about $\frac{1}{5}$ of an inch in length, much in shape like an ordinary snake with a rounded head and very pointed tail, and it wriggles about in the blood stream like an eel. The mature form of the parasite has not been very thoroughly identified. Bancroft and Lewis believe they observed it in the connective tissue in cases of elephantiasis. However, this is not quite certain, but in its immature form the creature has been frequently observed. It is found in tropical countries, both in the New and Old Worlds, and the form that we are speaking of has the peculiar property of living at night, being present abundantly in the blood at night and not appearing during the day. Other varieties of this entozoon have been discovered in which the habits seem to be diurnal, they abound in the blood-stream during the day and disappear from it at night; but the variety associated with haematuria is the nocturnal filaria. It seems to be distributed by a certain variety of mosquito. The mosquito bites a person—the subject of filaria—during the night, and absorbs perhaps in a small drop of blood a couple of hundred of these organisms. The mosquitos lay their eggs in water, then die and the filaria are set free. The water is afterwards drunk, and in this way the parasites become transferred to the human body, where they develop the creature which infests the blood. The filaria, in addition to the associated haematuria, produces blocking of the lymph channels in some cases, and elephantiasis arabum, in some great swelling of the scrotum, and in others chylous dropsy of the tunica vaginalis, and we may have alternating attacks of bloody and chylous urine.

These are the chief remarks I have to make in connection with the different causes of bleeding from the genito-urinary tract.

**A CLINICAL LECTURE
ON
ARREST OF PULMONARY TUBER-
CULOSIS.**

By DR. THEODORE WILLIAMS,

AT

The Hospital for Consumption and Diseases of the Chest,
Brompton.

Delivered on the 6th May, 1896.

GENTLEMEN,—I propose to say a few words to-day on the arrest of pulmonary tuberculosis. It is well now and then to put this subject forward, because some have questioned the efficacy of any treatment for phthisis. They say it goes through one course, and ends in one way, forgetting that tubercle runs through a kind of evolution. The bacillus sets up irritation inducing proliferation of cells, the formation of giant cells, and causing various symptoms and changes in the lungs, partly constructive and partly conservative,—that is to say, it induces on the part of the lung tissue an effort to isolate the attack, or get rid of the bacilli altogether. This part of the process is what I want to draw your attention to to-day. I want to show you how it is that nature does her very best to prevent the enemy in the shape of the tubercle bacillus from effecting a lodgment, and failing that, to isolate and limit the attacks as far as possible. Take the simple form first of all, that is miliary tuberculosis of the lungs, where you have a few alveoli attacked by the tubercle bacillus, which sets up a certain amount of proliferation of cells. Supposing the attack is not on a large scale, there is time for the human being, or rather the constitution, to limit the amount of mischief by throwing out what may be termed certain fortifications. The first fortification thrown out by the lung tissue is the giant cell; many people regard this cell as one of the numerous forms of phagocytes, but we all know what is meant by a giant cell, and this is what takes place. The giant cell comes into relation with the tubercle bacillus, and envelops it, and a fight begins; and if the giant cell wins, the tubercle bacillus is destroyed. If, on the other hand, the tubercle bacillus wins, the giant cell vanishes. Taking the case where the giant

cell is the winner, what happens is this:—The processes of the giant cell join with the processes of the next giant cell, and this goes on, and in time the whole mass is converted into fibroid tissue, which when carefully examined is found to contain no tubercle bacillus. That fibroid tissue is the basis of nature's resistance to the tubercle bacillus, and it is really the first element in the arrest of pulmonary tuberculosis. In any way we can aid nature to promote the formation of fibrous tissue in a consumptive patient, in that way we may hope to arrest the disease. Every one used to think at one time that people must die from tubercle, but we have found out from post-mortem examinations, more particularly in Paris and London, that a large number of persons who died from other causes when examined showed in their lungs the evidence of old tubercle. What is the proof of old tubercle in the lungs?

In these cases where the subject has died not of consumption but of some other disease, in a typical instance showing cured consumption, you find on opening the thorax, one or both lungs with a somewhat puckered look about the apex, and before you cut the pleura it feels as if there was some scar tissue below. You open the apex and find generally a few masses of fibroid tissue, almost always dark coloured, and so tough that, as the French say, it cries under the knife. I draw your attention to that (1) form, and I shall give you later on the clinical signs that it gives rise to. That is the simplest form—limited miliary tuberculosis, and occurring at the apex. Conversion takes place into fibroid material, and the patient is practically free from danger, for from that material there is no chance of auto-infection. Another common form (2) is where you have caseation and cretification, the cretified material being of course a degeneration of the caseous material, but surrounded by a tough envelope of fibroid tissue. Generally speaking, there is no communication between this mass and any bronchus, the bronchus, which previously led to the former cavity, having become blocked.

Encapsulation is of the greatest importance, and cases are recorded where these encapsulated caseous masses have existed in patients' lungs, and the disease has been perfectly quiescent for years, when owing to some injury or strain, or some accident, quite suddenly the patients have developed

active tubercular symptoms, and have died of tubercular meningitis or acute tuberculosis of the lungs, and after death it has been found that these patients have lived so long because the capsules remained intact. The capsule breaking has been the cause of the mischief, the caseous material escaping and becoming absorbed has caused a general infection. All this shows how important encapsulation is. As regards these caseous masses, of course they are as a rule dangerous, yet after a while they sometimes pass into a condition which may be described as safe. After a long period then, even if the capsule be broken through, infection may not always follow. Dr. Sidney Martin has made some experiments which point strongly in that direction: he took a number of these caseous nodules and inoculated guinea-pigs, without producing any result. It must be remembered that the slightest amount of tubercular matter is highly infectious when inoculated, into guinea-pigs,—animals peculiarly liable to infection by the tubercle bacillus. This showed practically, so far as these caseous nodules are concerned, that they may pass into such a condition that they cease to have the power of infectivity. A cretified mass at the apex proves that tubercle had been there, but it is not common to find these caseous masses at the apex: they are more common in the bronchial glands. The caseous material seems to be absorbed by the glands in the ordinary way, and then it remains there, and undergoing degeneration becomes a cretified mass in the gland. A favourable place to find these affected glands is near the root of the lung, and often you will not find any actually in the lung.

If you find cretaceous material in a bronchial gland at a post-mortem, you may be quite sure that tubercle has also been present in the lung somewhere. The third form (3), and the commonest form of all, is that of a contracting cavity, situated generally in the upper lobe, where is found the remains of a cavity usually with a very hard fibroid wall and a certain amount of puckering. In the specimen I now show there are a number of small cavities which have been obliterated by this fibroid material. You may find cavities partially contracted containing sometimes caseous material, sometimes a smooth lining, but in all these cases of real contraction you find the bronchus blocked

altogether, and that is why in life there are no signs of cavity to be found. This next specimen shows the puckering which occurs in the lung surface over a cavity. Another of the changes is the thickening of the interlobular septa, which become thick, hard, fibroid, and deeply pigmented, and being increased in size they show well the connection with the pleura. The interlobular septa are immensely thickened as a rule.

The changes in the pleura will next occupy our attention. First, in the simplest form, the localized miliary tubercle; all you have is a puckering of the surface of the pleura, and sometimes the effusion of a small amount of lymph, causing slight dry pleurisy, but in a large number of cases you get a number of small delicate fibrils passing from the parietal pleura to the pulmonary pleura, and the interstices of this sometimes get filled up with a certain amount of fluid. Another form is where you have a thickened pleura, as seen in this specimen I now show you, and the last is where pulmonary emphysema surrounds the apex owing to the alveoli being blocked; the alveoli, that are clear, enlarge from the pressure being concentrated on them, and bullæ form at the apex. It is only where the tubercle exists that this compensatory emphysema occurs, and it is scarcely found elsewhere, so, if you find these lesions in the lungs, you may be quite sure there has been arrest of pulmonary tuberculosis. The late Dr. Moxon said that fibrosis was the past tense of tubercle, and if you find fibrosis at the apex and nowhere else, you may be quite sure that it is converted tubercle, but you may find that the entire lung is fibrosed, and that may be from pneumonia or pleurisy, for these may cause fibrosis of the lungs. Fibrosis, however, plays a large part in the arrest of tubercle. These specimens I now show you by the kindness of Dr. Habershon demonstrate this point. This specimen is a case of cavity, the lung is fibrosed, some of the interlobular growth is increased, the glands are deeply pigmented, and at the apex there is an old cavity, the pleura is thickened and there is no bronchus opening into it. The subject had phthisis, and came in here and died not of consumption but of typhoid fever. At the post-mortem examination they found this state of the lungs, but the patient had had no symptoms of tubercle for years, that was all a

thing of the past, and therefore, without the history, this specimen would not be of much interest.

The next specimen is that of a thickened lung quite dark in colour, with a small cavity in the upper part; you can just see the puckering all round, and there is no bronchus opening into it. There is more than one cavity, and all are more or less contracted. In this case the lung has become fibrotic, and the cavities you will see are lined.

Let us consider what is more interesting, what will be the clinical evidence of a case of pulmonary tuberculosis undergoing arrest. We must take it in its various stages, though it is somewhat old-fashioned nowadays to talk of stages.

By the first stage I mean tubercle without any breaking down. Supposing a patient is undergoing arrest of the disease, the symptoms are very simple in the first stage; he is sure to be gaining weight, and probably improving in colour and general condition. But one thing is always to be looked out for, and that is in connection with the breathing. Supposing you have a patient come to you complaining that his breathing is becoming shorter, and you can find no reason for it from the physical signs, you can be pretty well certain that fibrosis is going on. If his breath is getting shorter, therefore, it may be a good sign. If I have a patient coming to me and saying that his breath is getting short, I am always glad to hear that unless the physical signs are adverse. The breath becomes shorter, the cough lessens, and in time the expectoration disappears altogether, and then the cough ceases and the patient gets well. On examining a patient of this sort you find a certain amount of dulness but no crepitaculum, but after a while in some cases you may find even of that dulness no traces whatever. That is because of the formation of emphysema at the apex, especially if much exercise be taken, otherwise you might imagine that the tubercular mass has been absorbed; but that is not so, it is not absorbed, it is there as a hard nodule, but the symptoms are masked by the emphysema. You find this very often occurring in those patients who have been sent to winter in high altitudes. I have often sent such patients away with well-marked dulness, bronchophony, crepitaculum, and a certain amount of tuberculous expectoration, and when they have

come back I have had to look up my notes to find which lung was affected. No doubt fibrosis has occurred, and emphysema has subsequently masked the lesion.

If you detect anything of that sort in a case in the first stage of phthisis, it is because emphysema has occurred, and you may be sure that arrest of the tuberculous process is taking place. Now when you have cavities in a patient's lungs and arrest of disease taking place, what happens is this:—First the cavity contracts, weight is gained, and that in a most remarkable manner; I found in two of my cases that the patients gained a stone a month, and actual contraction of the cavities took place in one month. We watched these cases here with interest; and it was the gaining of weight that drew our attention at first to the lung changes which were going on, and another point noted was that expectoration was diminished, and became offensive,—part of the cavity gets almost closed, and in the lower part the secretion is retained, and when it is coughed up it is offensive. The cough changes, and becomes more metallic, it is more tearing and violent, and what is described as frame-shaking, and the breath is always shorter.

Supposing you have cavities on the right side of the chest, and these changes take place, the cavities contract, and the first thing you notice is that the cavernous sounds are not so audible as they were, and where there was dulness, say in the first and second spaces, for instance, near the sternum, you get a line of resonance appearing (and I will show you presently patients where the line of resonance crosses the central line), you cease after a while to hear the cavernous breathing, or you will hear it well marked above the clavicle, and then when that ceases, above the scapula, and, after a while when that ceases, cavernous sounds may be found only in the interscapular region; these are instances of the extreme vagrancy of sounds, but I have seen each. So you see it is perplexing when you look for an old cavity and the signs have all gone, but it is thus explained by the lung contracting and fibrosis occurring. The contraction takes place always towards the fixed spot, and that spot is, if there be no strong adhesion, the root of the lung, and so the sounds follow that line, and you hear them gradually fading away as you leave the

fixed point. The cavity is contracted towards that fixed point, the lung is drawn away from the heart and to the back of the thorax, and you have the opposite lung spreading out, and therefore you hear in place of the cavernous sounds good vesicular breathing. If the cavity is on the right side, other changes take place, namely, dislocation of the organs,—the liver is drawn up, the liver dulness is sometimes right up to the fourth rib, the heart is drawn across, and you hear the heart-sounds under the sternum. Sometimes you can feel the heart's apex in the third or the fourth space on the right side of the sternum. The diaphragm is, of course, drawn up, and there are general changes of course on both sides, flattening of the chest, while there is diminution in the size of the chest,—one or two inches difference in the circumference. If you have a cavity contracting on the left side, the first thing you notice is that the movements of the heart are visible in the first, second, third, and fourth interspaces; the heart becomes more superficial, and the lung having been withdrawn from over it, the heart approaches close to the walls of the chest. The impulse of the heart, which is normally in the fifth space, an inch to the right of the nipple, you notice has moved towards the axilla, and you find it sometimes even in the axilla itself, and this may give rise to a murmur, which is due, as far as we can make out to the twisting of the aorta, from this displacement of the heart towards the axilla. In post-mortem examination no other reason for this change has been observed. The right lung is drawn across, the area of resonance traverses the median line, and the cavernous sounds gradually disappear in the way I have described on the right side, and the general course of arrest is thus indicated. In the first stage one of the earliest signs of the arrest of the disease is that there is a slight retraction or depression in the first space, and there is one feature that I have not alluded to yet, only occurring in very extreme cases,—the chest walls may fall in, the shoulder may become depressed, and spinal curvature may be well marked towards the affected side. I remember well a case that I showed at the Clinical Society of a man who had a tinkling cavity which had contracted, and in this case the contraction was very considerable, and the expectoration had diminished, but the

spine became quite curved towards the affected side, the shoulder was depressed, and there was a great sinking in of the chest wall; the patient was very short of breath, of course, but otherwise improved. What is the after history of these cases? In most patients with contracting cavities when the dyspnoea arising from so much lung fibrosis has become chronic, it is found that they are emphysematous to a great extent, they are short of breath, and they become more or less bronchitic, and they are very often known as chronic asthma and chronic bronchitic cases, and as a rule nothing more in the matter of tubercle is looked for or thought of. A man of 60 with a hyperresonant chest and shortness of breath is often diagnosed as asthmatic or bronchitic, but if in any of these cases after death there is a post-mortem examination, in the apices of the lungs there will be found undoubted remains of old cavities, showing that the patients have suffered from tubercle in the course of their life, and that arrest of the tubercular process has occurred. These patients, then, may live on to old age, but in some instances fibrosis of the lungs once started persists, and that is a serious matter because these people get more and more short-winded, and the fibrosis attacking the lung very considerably, there is a great obstruction of the vessels of the lung and all sorts of trouble may arise. The circulation is blocked, the heart becomes dilated, oedema occurs in the legs, and after a while becomes general dropsy; then from the tax on the kidneys renal disease arises, and these cases die from the results of obstruction of the circulation and respiration. What is the prognosis in these patients? It all depends on how much of the lung is originally involved; if an upper lobe only is involved, and there is not much affection of the rest of the lung, they may live for the natural term of life, but they will always be short of breath, and it is to be noted that in only a few of these cases does fresh tubercle form. Where there is very extensive tubercular infiltration of the lungs, and only a small amount of fibrosis, the tubercular inflammation may easily be again lit up, and in these cases the prognosis is bad, for they have no power of resistance against the disease, and this determines the progress of the case on the downward path. Many of these patients in which the arrest of the tubercular

process has occurred, are as you know very short-winded, but there is many a judge now sitting on the Bench, there is many a barrister now pleading his cases in the courts, and many a clergyman preaching eloquent sermons, many a medical man in active practice, who to my knowledge have at some time or other suffered from tubercle and recovered. If you go into the personal history of the lives of some of our eminent statesmen you will find that they have suffered from blood-spitting, and you may find that they have been exiled from their own country for a time on account of their health. I have to-day taken, of course, a very hopeful side of the question, my lecture being on the arrest of pulmonary tuberculosis and on nothing else, though of course I am quite ready to admit that the cases I have been discussing are not always the rule, and there are, unfortunately, many that go to the bad; but as regards the proportion it has been found from the post-mortem records at several hospitals that the proportion of old tubercular changes found in the lungs of subjects dying from causes other than tubercle is about four to nine per cent., some give even higher percentages.

Patients exhibiting the various forms of arrest of pulmonary tuberculosis were exhibited.

A THURSDAY CONSULTATION AT ST. BARTHOLOMEW'S HOSPITAL.

Dislocation of the Elbow.

Mr. HOWARD MARSH: This I am told is an obscure case of injury at the elbow; I have not till now had the opportunity of seeing the case, and the history is not a very satisfactory one as to the cause. I am inclined after examination to consider that it is a simple dislocation of the head of the radius, which has slipped out of the orbicular ligament. This is an accident which has been very clearly described and definitely established, and the way in which it has usually been produced is that the child has had its hand pulled with a sudden jerk forward of the wrist, and I think you can understand that if the hand has been violently in this way pulled forward, the radius following it down may slip out of the orbicular ligament. When you are called

upon to examine an injury of an elbow in a child, you are very likely to find that some fracture has taken place at the lower end of the humerus. Here, however, the two condyles are in their normal position, the olecranon is in its normal situation, and otherwise there is no evidence of the presence of callus, or any change of the outlines of the lower end of the humerus. When these cases of dislocation are recent, surgeons have succeeded in reducing the displacement, manipulating the head of the radius back into the ring of the orbicular ligament; but I should despair of doing that after two months have elapsed since the injury. It is almost certain that the parts have been very much altered. Then the question naturally arises whether it would be advisable to undertake any operation for the sake of replacing the head of the bone in its proper relation; I am not aware that that has ever been done. I know in these cases, however, that the usefulness of the limb is but slightly impaired. The forearm, you notice, has full power of supination and pronation. In the cases I have had an opportunity of examining and seeing, I have noticed that the functional activity of the limb has not been materially impaired. For my own part, I should be inclined to say that the elbow had better be left alone.

Mr. LANGTON: I think one must exclude any damage here to the lower end of the humerus or separation of epiphyses at the joint. The ulna also is in its right relationship. I think, therefore, that as the ulna and the humerus are in their normal positions, the disease may be limited and localised to the head of the radius. It is not quite the usual displacement which has here taken place. The displacement which usually takes place lies as a rule more in front of the condyle. There seems to be a little bowing of the radius, where there may be, in addition to the dislocation of the bone, some separation of the epiphyses which has united. I am certain that it would be better to let this case alone. I have seen one case in which an open operation was performed for a similar condition, which was followed by the ultimate destruction of the elbow-joint. In many other cases left without operation the resulting movement has been quite satisfactory, leaving only a deformity. It does not much matter about the appearance in this case, the patient being of the male sex.

Mr. BURLIN: I take the same view as Mr. Marsh, and think it ought not to be operated on.

Mr. WALSHAM: I think there is a dislocation here, but it struck me that there was some fracture of the ulna a little below the joint; there is a suspicious lump there that might well be callus. I take Mr. Marsh's view that a sudden pull on the hand might displace the bone from the orbicular ligament, especially if a fracture of the ulna occurred at the same time, as this would allow of a stronger pull on the head of the radius. I admit that there might be some risk in cutting down on the bone, but there could be very little with the case in the hands of Mr. Marsh; and on the whole I should explore the condition by an operation, and try and get the bone back into its place.

Mr. HARRISON CRIPPS: I quite agree with the diagnosis that has been expressed. So far as treatment is concerned, I would suggest that a thorough trial be made under an anæsthetic to reduce the dislocation. If this should fail owing to the length of time since the dislocation, I would subsequently see what could be done by an open operation. I should not immediately proceed to operation even if one could not reduce the dislocation. I should go on if necessary to an open operation afterwards.

Mr. BOWLBY: The radius is displaced outwards, and I do not make out any fracture. I should make an attempt to reduce the dislocation, and quite agree with Mr. Walsham that no harm should come of an operation, and some good certainly might.

Mr. HOWARD MARSH: I feel that it would be worth while to manipulate the joint under an anæsthetic, to try and get the head of the bone back into position. On the whole I think the case should be left for a time to see how it goes on, and perhaps if any material defect remains, some operative interference may be considered.

Hypertrophic Morphea.

Mr. HARRISON CRIPPS: This next case is a patient, 13 years of age, who is said to have had this growth you can see on the right side of the face when he was born, but latterly it has greatly increased. When I first saw it I thought it was an ordinary case of lupus; but when I heard the history, that he had had it as a congenital con-

dition which had been slowly increasing for years, I thought it could not be tubercular. There is no real granulation tissue, only simple tissue in which you can see the vessels ramifying in a way you cannot see in lupus; there is nothing like tubercular ulceration, and I do not think that congenital lupus is known. It reminds me of a case which was shown here by Mr. Langton a short time ago, and that particular case I regarded as one of the forms of morphea, or keloid of Addison. This keloid shows itself in many ways, and begins in an hypertrophied condition of a patch of skin, and slowly extends; it may be pigmented and brown as in the case before us, and after a time it increases, spreading from the centre, there is no ulceration, the central portion clears up, leaving a clear skin, and that is the condition we have in this case before us now. As regards the treatment, local treatment is of not much use short of excision; my intention is to cut round that keloid, giving a clear margin of about one eighth of an inch.

Mr. LANGTON: At first sight it looks tuberculous, but it certainly has many features in common with the case I showed here the other day. I excised the growths in that case the other day, and I have not heard yet from the microscopists whether they are quite sure what it was, whether it was an inflammatory or a new growth. There were in that man's back several other nodules, I think there were three altogether which I also excised, one of them lying some distance from the other lumps, which I excised separately. We are still awaiting the decision of the gentlemen to whom the specimen was sent to be microscoped. I should deal with this case before us now in the same manner as I dealt with that case of mine.

Mr. HOWARD MARSH: I should have thought at first that it was lupus, but I quite bow to your opinion and follow your diagnosis, and certainly agree with your treatment.

Mr. WALSHAM: I also agree with what has been said in regard to this case.

Mr. BOWLBY: This condition is said to have begun congenitally; I do not know that morphea or lupus are generally recognised as congenital affections. I may say in regard to Mr. Langton's case, so far as the microscopical examination is concerned I should say that it was certainly inflammatory. I am speaking of course of the case that was under the care of Mr. Langton, but

I should regard this case also as inflammatory. As to treatment, when you do the operation you might cover the area with some Thiersch's skin grafting.

Mr. HARRISON CRIPPS: I used the word morphœa merely as a name; it conveys no explanation as to the cause of the growth: certainly after what has been said I shall excise the growth.

Syphilitic Tongue becoming Epitheliomatous.

Mr. THOMAS SMITH: The case I show you now is that of a man 61 years of age. Twenty-one years ago he had syphilis. Ten years ago there was ulceration of the tongue, which gradually got well. Six years ago there was more ulceration of the tongue, which also got well. Three years ago there was another attack, which got well under iodide of potassium. In January, 1896, he noticed a patch on his tongue at the back on the right side; and here there is a soft papillomatous growth; in front more recently there has come a patch, with a deep excavated ulcer with hard edges. The tongue is fissured all over. The ulcer near the tip only came six weeks ago, and there is no pain in connection with this ulcer. This is a typical syphilitic tongue which has had constant irritation off and on for twenty years, and one part in front has become cancerous. It looks as if it were epithelioma, the edges are hard, and under the chin there is to be felt a round hard lymphatic gland. I believe him to have an epitheliomatous growth, which is very commonly grafted on to tertiary syphilis. The question is, shall I continue to treat him with iodide of potassium? He says his tongue gives him no pain, and that is rather an unusual circumstance. Perhaps the best thing would be to keep him a little longer under iodide of potassium, and afterwards excise a considerable portion of the tongue with the ulcer in front, and the papillomatous area on the right side.

Mr. LANGTON: There can be no doubt that it is syphilitic; whether there is any disease beyond grafted upon it appears to me a matter for consideration. I should think the ulcer at the tip is probably gummatous in its origin, but it may have taken on the form of squamous epithelioma; the enlarged gland under the symphysis would rather, I think, point to that, for a tertiary gumma breaking down would not lead to lymphatic glands becom-

ing infiltrated. The absence of pain is rather in favour of the idea that it is gummatous and not squamous epithelioma, for in squamous epithelioma pain is generally one of the most prominent symptoms. There is, perhaps, some squamous epithelioma there, but I should be inclined to give him large doses of iodide of potassium and a little mercury, and then watch the effect.

Mr. HOWARD MARSH: I should think that here, as is so common, a change has taken place from syphilis to epithelioma. I should think that the gland points strongly to that change having happened. I think it is a little doubtful, however, and you might treat him for a short time with iodide, and see if there is any increase of induration, and see if any other symptoms arise which would necessitate an operation.

Mr. BUTLIN: It is an old syphilitic tongue, but I do not feel sure if the ulceration at the tip is epitheliomatous or gummatous. The diseased part of which I have little doubt is the warty growth on the right border. I believe it to be epitheliomatous; if not now, at any rate it will be so in a very short time. My feeling would be to remove that tongue at once, and take out the gland. If there was still any doubt remaining, I should cut off a portion for examination under the microscope, and you would know soon exactly what it is.

Mr. THOMAS SMITH: You think I should take the whole tongue out?

Mr. BUTLIN: Yes; removal of the whole tongue is required.

Mr. WALSHAM: I see no great objection to iodide of potassium being used for a short time longer, though I think it is a case of squamous epithelioma supervening on the top of the syphilitic affection, and I should not wait long, but if rapid improvement does not take place remove the whole tongue.

Mr. HARRISON CRIPPS: I think it malignant disease, and I should excise the tongue.

Mr. BOWLY: I think it is epitheliomatous, and under any circumstances I should advise the removal of the tongue.

Mr. THOMAS SMITH: As regards iodide of potassium, I once had a very curious thing occur in my practice. I once had a patient to whom I gave a very considerable quantity of iodide of potassium before removing his tongue. After I had removed the tongue, the stump swelled up and filled the mouth and protruded from his lips, and I attributed it to the iodide of potassium I had been giving him. I am, therefore, now very reluctant to undertake any operation while the patient is taking iodide of potassium. I shall put all the facts of the case before the man, and he will have to decide for himself what shall be done.

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CLINICAL LECTURE ON THE DIAGNOSIS OF CIRRHOSIS OF THE LIVER.

Delivered at the Central London Sick Asylum, in connection with the London Post-Graduate Course, May 14, 1896,

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LADIES AND GENTLEMEN,—I have chosen as the subject of my lecture to-day: "The diagnosis of cirrhosis of the liver." As you are aware, cirrhosis of the liver is exceedingly common, and in many cases its recognition presents no difficulties; but, on the other hand, though common and well-known to us, it often does give rise to considerable doubts and difficulties in diagnosis. Thanks to Mr. Hopkins, we have a series of cases here which illustrate some of the points which occasion doubt at the bedside.

The term "cirrhosis of the liver" should be restricted to the cases in which there is a marked overgrowth of connective tissue more or less uniformly distributed throughout the liver, which, by contracting, obstructs the radicles of the portal vein and compresses the hepatic cells. I make this definition because there is a tendency to regard other conditions which give rise to contraction of the liver and portal obstruction as cirrhosis. I think it is better, in the interests of pathology, to restrict the term to cases where the conditions I have mentioned are present.

Cirrhosis of the liver is almost invariably due to the influence of alcohol in excess. I say "almost invariably" because, as is known, we do meet with cases where there is no history of such indulgence,—as, for instance, in children. But even here it is a well-known fact that some cases of cirrhosis in children have been clearly traced to alcohol, so

that the fact of our patient being a child should not mislead us, nor cause us to dispense with a careful inquiry as to habits. Some cirrhotic children have been quite addicted to the consumption of spirits. I show you the drawing of the liver from a case which I showed at a discussion at the Pathological Society some years ago—a little girl who was under my care at the London Hospital, who came with considerable swelling of the liver, and in whom the fatal termination was caused by haematemesis. That little girl had practically never tasted alcohol, so that cirrhosis may occur independently of it. The late Dr. Moxon suggested that in some of these cases inflammation may spread to the liver from the peritoneum, and one or two cases have been traced to that cause.

Cirrhosis generally occurs in those who "nip," rather than in topers, but you will meet with examples of both. You must also bear in mind that the amount of alcohol taken acts differently on different people, and their susceptibility varies; moreover, the statements made by patients are not always trustworthy. The next point to remember is that in cirrhosis the liver may become enlarged and subsequently contract, or it may remain enlarged until death, or there may be no evidence that the liver was at any time larger than natural; and it may become very contracted.

But the object of my lecture is not to discuss the pathology of cirrhosis of the liver, but its clinical recognition. The diagnosis has to be made in two stages—the early stage, before the signs of portal obstruction are at all complete, and the later, when the portal obstruction is greater and has given rise to ascites.

In the first stage the diagnosis is notoriously extremely difficult, and we really, in the majority of cases, suspect it rather than are able to demonstrate it. The reasons for suspecting it are: the undoubted fact of the patient's over-indulgence in alcohol, and the occurrence of dyspepsia of an alcoholic character—anorexia, pain after food, morning vomiting, morning diarrhoea, and general irregularity of the bowels.

Accompanying these symptoms there is often

the first evidence of portal obstruction, viz. the occurrence of haemorrhoids, also pain in the region of the liver. In addition, the *facies* of the patient and the odour of the breath furnish contributory evidence. If there is the earthy, sallow aspect of the cheeks, and dilated veins on the same part, with a slight degree of icteric pigmentation, which all point to some hepatic disorder, especially if the urine be scanty and turbid. Such patients generally waste, because when such an important organ as the liver is diseased, metabolism is interfered with and nutrition is perverted. Another reason is that the taking of alcohol diminishes the normal appetite for food. In this stage, before there is any very marked portal obstruction, we do sometimes find undoubted physical evidence of the existence of cirrhosis. In some cases the liver is abnormally small; in other cases the liver is enlarged, but the portal obstruction is not sufficient to produce ascites. A publican came to see me quite recently whose liver reached from the sixth rib to the umbilicus. There were great tenderness in the epigastrium, injected venules of face, his urine contained bile, and the liver was tender on pressure. The tenderness in the epigastrium was probably due to gastritis. I diagnosed cirrhosis of the liver, and warned the patient's mother that haematemesis would very likely occur. A few weeks after this the patient had a severe attack of haematemesis (I saw him in consultation with his medical attendant), and he got somewhat better for a time. Then he had severe and persistent hiccough, which lasted two or three weeks in spite of treatment. Last of all he had a renewal of the haematemesis, which brought about a fatal termination. We have in a bed here a patient who has enlargement of the liver, no doubt due to cirrhosis, where there has been no ascites, and is none at the present time. That patient also has phthisis, an association which is not by any means uncommon.

I now pass to the diagnosis of cirrhosis of the liver in its second stage, namely, when the portal congestion is more advanced and leads to ascites. In some of these cases the diagnosis is easy, in others it is difficult. We may approach the diagnosis from various points. First of all, we may take the occurrence of ascites as our starting point, and consider the various causes which give rise to it, and, by excluding all others, prove that it is due to cirrhosis. That is a process which is

very commonly adopted; a patient comes with an enormous collection of fluid in the abdomen, and the most prominent symptom is taken as a basis for the solution of the problem. Of course, ladies and gentlemen such as I see before me do not need reminding of the necessity for making sure that the collection of fluid is really a peritoneal effusion—that it is not an ovarian cyst, for instance. Having ascertained that there is ascites, we proceed to consider the various causes which must be borne in mind. As in the publican whose case I have narrated, the cirrhosis may pursue its course to a fatal termination without the occurrence of ascites at all. Sometimes, preceding ascites there is great distension of the abdomen, (a point on which the late Dr. Sutton used to lay stress,) and when that is everywhere resonant from distension of the intestines with gas, the advent of ascites may often be suspected. Probably the weakened condition of the intestines is the cause of that.

Well, *dropsey* beginning in the peritoneum points to a local origin, and suggests obstruction of the *vena porta*. Of all such causes, cirrhosis is the most common. But other causes must be excluded before we can be positive, and of these other causes, *syphilitic contraction of the liver* is one of the most important. It is sometimes termed syphilitic cirrhosis, a name I do not use, believing it wiser to abstain from using the word cirrhosis in connection with syphilis, because the prognosis and treatment of the two diseases differ widely. In cases of syphilitic contraction of the liver we inquire as to the history of syphilis, and look for evidences of the existing disease, namely, nodes, eruptions, ulcerations, etc., or of past disease, such as the scars of previous ulcers or of suppurating nodes, cicatrices in the palate and throat, iritis, choroiditis, etc. We have in the ward here a woman who has an enlarged liver without ascites, in whom you will see undoubted evidence of syphilis at the present time. She has been under Mr. Hopkins' care for some years. Though she has not yet got ascites, we know that her condition may produce it, so that the case presents an instance of the difficulty which some cases present of distinguishing between syphilitic contraction and cirrhosis.

The next cause of obstruction of the portal vein is *perihepatitis*. This is a curious condition in which the peritoneum over the liver becomes enormously thickened. Sometimes it is called

capsular hepatitis, and the liver looks as if coated with a layer of lard, which layer may be an eighth, a quarter, or nearly half an inch thick, covering the liver uniformly. This goes on contracting and interfering with the circulation of the blood through the liver, giving rise to signs of portal obstruction. With it, there is usually a similar inflammation of the spleen, and very often thickening of the peritoneum generally. These cases are very difficult to distinguish from cirrhosis,—indeed, in many such distinction is impossible; they are recognized as cases of portal obstruction, and cirrhosis is diagnosed. Though the distinction is not clinically important, the pathological process is somewhat different. It may be suspected in cases of contraction of the liver where there is no evidence of alcoholic excess in the patient's history or appearance, and by attacks of pain in the liver of a more severe character than is usual in cirrhosis.

The next condition which may simulate cirrhosis when there is ascites is the occurrence of *tumours in the abdomen*, including carcinoma. Here a tumour is usually to be felt, or an enlargement of the liver with irregular projections on the surface. In cirrhosis also, as you are aware, the liver often feels uneven, but in carcinoma the projections are larger and coarser. These tumours pressing on the portal vein do undoubtedly give rise to great difficulties in diagnosis, but these difficulties are usually in the direction of mistaking cirrhosis for carcinoma rather than the converse. Pain, in the case of carcinoma, is generally much more persistent and enduring than in cirrhosis, and in these cases there has usually been a history of wasting for some considerable period before the onset of the physical signs. We have here a patient with a liver enlarged and with masses in it which can be easily felt, but who does not quite present a condition which best illustrates the difficulties, because he has no ascites. In many cases carcinoma of the liver gives rise not only to ascites but also to jaundice. The occurrence of marked, persistent jaundice with ascites should cause you to suspect the occurrence of carcinoma.

Then, *thrombosis of the portal vein* has to be considered. Cases occasionally occur in which thrombosis takes place quietly, and the vein becomes entirely obliterated by the persistent portal obstruction. This, accompanied by ascites, makes

a correct diagnosis during life, as a rule, impossible. Of course where very rapid thrombosis occurs no confusion should result, because then the train of symptoms is very different. Ascites develops in one or two days, with great dilatation of the superficial veins, and very often with the occurrence of haemorrhages—striking conditions which make the diagnosis fairly easy.

Another condition which may be mistaken for cirrhosis is *cancerous tubercle*, or miliary cancer of the peritoneum. This disease is practically always secondary to some primary focus of carcinoma in one of the abdominal organs (though it may be latent). Miliary carcinoma of the peritoneum is usually, as far as my own experience goes, confined to adults; it sometimes runs a non-febrile course, and gives rise to much ascites and great emaciation. I have known this condition mistaken, on several occasions, for cirrhosis. The absence of a history of alcoholism, and the discovery of enlarged glands in the groin, are important points; and, a more rare concomitant, the occurrence of subcutaneous cancerous nodules over the abdomen, spreading thence to other parts, have in some cases enabled a correct diagnosis to be made.

Tubercular peritonitis in the adult is occasionally a cause of mistake. In these cases the omentum becomes doubled up into a hard mass, which may be felt in the upper part of the abdomen, and may simulate an enlarged liver. The history, in some cases apart from evidences of drink, and the co-existence of tubercle in the lungs or elsewhere, are important guides. As regards the lump which may be felt in the abdomen, very often a line of resonance may be detected between it and the hepatic dulness.

Occasionally we find *contraction of the liver* in connection with *heart disease*. These cases are not so common as one would suppose from reading. I have seen one or two cases where, after long-standing heart-disease, dropsy, after subsiding, has recommenced in the abdomen, and where there have been ascites and some contraction of the liver. In a certain sense this is cirrhosis, but not alcoholic cirrhosis. I had a case under my care not long ago, in which the patient had dropsy in the legs, due to heart disease. The dropsy disappeared and he improved, and then about one year and a half after the subsidence of the dropsy,

ascites came on. The liver was found contracted, the primary cause of which was the heart disease.

Renal dropsy hardly ever gives rise to practical difficulty, though there may be large ascites. The dropsy begins in the face or hands, either antecedently to or coincidently with the ascitic collection, and the condition of the urine and cardiovascular changes enable the diagnosis to be made.

Having, then, excluded the various causes of ascites, and cirrhosis remaining the most probable cause, we look for confirmatory evidences. These are :—a history of alcoholic indulgence, and the signs of such—the complexion, facies, the occurrence of piles, &c. In such cases we may find the liver either small or large. We have two examples here: one a woman, in whom, with cirrhosis, the liver is small; and the other in which the liver is enlarged.

This brings me to speak of “hypertrophic cirrhosis.” The term is differently used by various writers; there is a lack of concurrence of view on the subject. By some writers and observers cirrhosis, with enlargement, is spoken of as hypertrophic cirrhosis. Others, on the other hand, limit the term to cases where the pathological condition differs from that of ordinary cirrhosis. The exact pathology of this condition remains still in dispute. It is believed by Charcot, Gombault, and others, who first drew attention to it, and they are confirmed by subsequent writers, that the disease starts from the bile-ducts, and that a disease of similar nature has been produced by tying the bile-duct in animals. It differs histologically in that the cellular infiltration which precedes the formation of fibrous tissue and leads to compression of the portal vein, is much more intra-lobular than interlobular; and in some cases it is spoken of as insular, *i. e.* nearly every lobule seems to be invaded, and the growth is almost pericellular. Not only does it surround individual lobules, but we find embryonic connective tissue between the individual cells of the lobules. Hypertrophic cirrhosis, as described by Charcot, is exceedingly rare, and it has certain features which are quite characteristic. In the first place, the liver is very much enlarged, generally more than in any other form of cirrhosis; livers weighing 6 lbs. and 5½ lbs. have been reported. Not only is the liver very much enlarged, but it is generally smooth on the surface, with a hard, sharp edge. The patient is deeply jaun-

diced, very often of a green colour, and an important point is that it is characterised, as a rule, by an absence of ascites. This form is apt to be attended with grave and fatal nervous symptoms; the patient sometimes sinks into a comatose condition, occasionally preceded or varied by convulsions; the temperature often becomes subnormal, and haemorrhages take place. The symptoms which occur in ordinary alcoholic cirrhosis in early stages may be present, but they most resemble the toxic or nervous symptoms which are often seen in acute yellow atrophy. On microscopic examination you see, in addition to the fibrous tissue of the character mentioned, very often long rows of cubical cells, which have been described by some as newly formed biliary canaliculi, but which others say are hepatic cells which have been displaced or deformed by the contraction of the fibrous tissue. It is stated by some writers that this form of biliary cirrhosis is not due to alcohol, and they would speak of it as like hypertrophic or biliary cirrhosis. My own experience is that it is associated with intemperance. One case made a strong impression on my mind, in a patient whose condition was diagnosed as carcinoma, as most of these cases are. It was believed to be carcinoma up to the time of the autopsy, and the inquiry as to alcoholic indulgence was probably not pushed as it would have been had cirrhosis been suspected. Afterwards a history of alcoholic indulgence was established beyond doubt. Therefore that is a very important point. Remember that hypertrophic cirrhosis, with deep jaundice and an absence of ascites, is very frequently mistaken for carcinoma; also that biliary cirrhosis is very uncommon, though it is loosely applied to alcoholic cirrhosis with enlargement.

We also find that there is a great difficulty in distinguishing between syphilitic disease of the liver and hypertrophic cirrhosis. Syphilis sometimes enlarges the liver to a very great extent, of which I have an example here from the London Hospital, under the care of Dr. Samuel Fenwick, who has kindly allowed me to show the patient. The patient had been under observation for about ten years when ascites commenced. He has not stinted himself in the matter of alcohol, but he had syphilis at the age of twenty, and there are some scars on the legs and elsewhere. He has an enormous liver, and very great dilatation of the

superficial veins. It was to demonstrate that condition that I had him brought here to-day. We meet with cases in which we have the greatest difficulty in distinguishing between syphilitic disease, carcinoma, and hypertrophic cirrhosis. Recently I had under my care a case sent me by Dr. Ash, of Kimberley. This gentleman had had several attacks of severe pain in the abdomen, and the case was regarded as biliary colic. It was noticed that his liver continued to enlarge, and when he arrived in England last March it reached to the umbilicus. He was intensely jaundiced, and suffered so much from pruritus that his life was a burden to him ; he was also very much wasted.

He continued exceedingly bad until a month or two ago, when Mr. Treves performed laparotomy. Mr. Treves found the liver slightly irregular and very much enlarged, presenting an appearance which did not enable him to decide its actual nature. He passed a probe along the common duct into the intestine, and completed the operation. For some time no bile passed into the intestine, the patient became exceedingly ill and emaciated, and seemed in peril of death, when quite gradually the bile began to pass into the intestine, the urine became gradually paler, he began to take food, and from that time (two months ago) he has steadily improved ; the jaundice has practically disappeared, he is gaining flesh quickly, and is passing normal, properly coloured motions. There were three possible diagnoses, all made by good observers—carcinoma, hypertrophic cirrhosis, and syphilitic disease. I did not gather from Mr. Treves that there was anything which indicated contracted fibrous tissue and great deformity which one sees in syphilitic contraction, so I think it was a case of hypertrophic cirrhosis, and that by the re-establishment of the natural passage of bile the symptoms have been ameliorated, and I hope the patient will recover.

We see cases of cirrhosis where the liver is large, but where there is no jaundice but marked ascites, cases which are often incorrectly called hypertrophic cirrhosis, and of which we have two examples here. Such cases do not present any difficulty. As a matter of fact, enlargement of the liver with cirrhosis is more common than contraction of that viscus. Only in a minority of cases is the liver found, in a fatal issue, in a contracted condition ; whether all cases would lead to contraction eventually I cannot say. Enlarged liver is specially apt

to occur in beer-drinkers, on account of the great increase of fibrous tissue, and the accumulation of fat in the peripheral portions of the lobules of the liver ; such cases do not present any great difficulties in diagnosis.

Then there is cirrhosis with contraction and ascites, which are not difficult to recognise. When there is an extreme degree of ascites, it may be impossible to determine the size and shape of the liver without paracentesis.

In all the conditions of portal obstruction which I have been speaking of, a collateral circulation must be established for the blood, and we see evidences of this in nearly all cases of cirrhosis. The patients who survive longest are those in whom the collateral circulation is most easily established. I need not now go into the various routes by which the blood may be carried away from the liver, but until that is done, the tendency must be for the pressure to remain in the portal system and for the occurrence of ascites. We see one or two cases in the ward of patients who have had ascites, have been repeatedly tapped, and in whom the fluid ceases to collect after a time. It is difficult to say why it is. The patient in No. 5 bed, who has been tapped repeatedly, tells me that after the last tapping his abdomen filled up just as on previous occasions, and tapping seemed again imminent when, one afternoon, he noticed he was passing more urine than before. That diuresis went on ; the fluid in his abdomen subsided about two months ago, and now he has only a moderate collection. Whether the increased passage of urine marked the exact time when the balance was reached, and the collateral circulation was established, I cannot say. Apparently nothing which he was taking induced the diuretic action, but increased urine occurred at the same time as the decrease in the abdominal fluid. Conversely, when patients have portal obstruction, we find the urine is scanty, and, as is usual with scanty urine, it is highly coloured and intensely acid, with a more or less copious deposit of urates. All the cases we have here are those in which this equilibrium has been reached, by one means or another, and when the ascitic accumulation goes on no longer. In that state a patient may go on for a considerable time, until very often cardiac failure leads to recurrence of dropsy, the patient breaking down under various

conditions which embarrass the heart's action ; ascites is then very apt to occur, and a fatal issue brought about.

We should take a very narrow view of the disease if we confined our attention to the liver. You must remember that practically always there are coincident changes in other parts of the system to which I need only briefly allude. Enlargement of the spleen is often of diagnostic value. None of our present cases happen to show that, but this, with contraction of the liver associated, is of the greatest value in diagnosis. Sometimes we get valuable aid from the implication of the nervous system. The patient in No. 6 has lost his knee-jerks, and two years ago he had weakness of the legs, which gradually passed off. There is every reason to believe that he has had peripheral neuritis, and if you are able to exclude other causes of peripheral neuritis, this disease will help you to confirm the diagnosis of alcoholic cirrhosis. Again, the kidneys are very prone to be involved. Granular contraction of the kidneys is not so common as you would expect ; the kidney is much more frequently large and white, a fact which was brought out very strongly by Dr. Dickinson in the alcoholic discussion at the Pathological Society, and in previous communications on the same subject.

Patients suffering from cirrhosis may reach the end of life in various ways. The most common is haematemesis ; this may sometimes persist for days together, and at the autopsy no source of haemorrhage can be discerned beyond the presence of the portal obstruction, or an oozing from the lower part of the cesophagus or from the stomach. Sometimes haematemesis is the first symptom to reveal cirrhosis. When this vomiting of blood occurs in an adult, the first question you would ask yourself would be : Is this a case of ulcer of the stomach, or is it cirrhosis ? In many such cases we find the liver is large, and there are other evidences of portal obstruction, which enable us to proceed to the correct diagnosis. Sometimes cerebral or toxic symptoms close the scene ; at other times, pneumonia or peritonitis. I think peritonitis was more common in olden days than it is now. When I commenced to study medicine we used to put off tapping as long as possible, and then used a very large trocar ; now-a-days we tap earlier, and more frequently, and use a smaller trocar. But

most important of all, we are careful to secure complete asepsis. It used to be taught that the tissues were very often subject to chronic inflammation, and this led many physicians to postpone paracentesis. We ought to tap without further delay when the breathing becomes embarrassed and the pulse small, not necessarily to empty the abdomen of fluid ; if we draw off a certain amount, very often the remainder continues to diminish, and the patient gets better for a time. I do not say that all cases of cirrhosis should be tapped. We should keep the patients in bed, and feed them properly, and then the ascites may go away without tapping.

Now, one of the most practical points, which has been impressed upon me over and over again, is, when making your diagnosis always ask yourselves, "Is this possibly syphilitic ?" I cannot exaggerate my feeling as to the importance of this. Syphilis may give rise to portal obstruction, and to ascites, and it may be difficult to draw a distinction ; but it is necessary, because the prognosis and treatment differ in the two cases. Syphilitic cases, as a rule, do better, and the collateral circulation is more easily established. In syphilitic disease the liver may be more deformed than in cirrhosis ; large chunks of it may appear almost cut off, but between those parts the organ may be quite healthy. These cases also last longer than cirrhosis, and collateral circulation is readily set up. I had one such patient with enormous ascites due to portal obstruction, who remained in good health for many years afterwards. At autopsies we frequently find livers which have large gummatous masses, much of which could have been got rid of by appropriate treatment, and the patient's life prolonged.

Let us now look at the patients. This man has syphilitic disease of the liver ; the organ is enormously enlarged, hard and irregular, and its edge can be felt all along. It projects upwards to the third rib. He has been tapped twenty-three times, and the obstruction of the portal vein has given rise to this very remarkable telangiectatic condition of the abdominal veins. These varicosities when once established, generally remain, but they are not necessarily evidence of the existence of severe portal obstruction at the present time ; you cannot infer whether the obstruction is in the portal vein, or in the inferior vena cava, because the blood is

so quiet and stagnant that it will flow where there is least resistance from time to time. He has been, as I have just said, tapped twenty-three times.

Here is a drawing of a still more extreme condition of obstruction. It was a primary carcinoma of the kidney. When the patient first came under care there was only portal obstruction, and these veins used to dip down and empty themselves into the groin. But later, the inferior vena cava became obstructed, and then all the blood from the lower extremities and from the portal circulation had to pass by these superficial vessels and empty themselves into the superior vena cava.

This young girl presents another syphilitic case. She has a greatly enlarged liver, its lower edge being below the umbilicus. She has no ascites, and no portal obstruction, being in the state before they occur. She has some nodes on one arm, and on the legs, and an ulcer on the palate, with extensive destruction of the soft palate.

The next case is one of cirrhosis of the liver with contraction. She has been tapped fifteen times. She has xanthoma, is subject to headache, but has never had jaundice.

This poor old man is suffering from carcinoma of the liver, not simulating cirrhosis. You can see, as he lies, a tumescence in the upper part of the abdomen, of an irregular character, and the whole moves with the descent of the diaphragm. None of the large masses press on the vena portæ; if they did he would have ascites. The physical signs, coupled with his appearance, makes the diagnosis only too easy.

Here we see cirrhosis with enlargement of the liver, but in the pre-ascitic stage. Up to the fifth rib there is dulness, and in this patient phthisis is associated. Bronchial breathing and crepitant râles can be distinctly heard at the apices.

The next patient presents a very typical case. He has a large liver, and dilatation of superficial veins, which indicate portal obstruction, though the diaphragmatic zone does not. This patient has been tapped fourteen times, and enormous quantities of fluid have been drawn off. He was about to be tapped the fifteenth time, when he began to notice that his urine flowed quite freely.

The last case is cirrhosis with contraction of the liver; his spleen can be felt, a very important combination.

You have now seen most of the phases in which

cirrhosis is generally encountered; but we have not a case where the ascites is very active, and masks the symptoms of contraction or enlargement.

A NOTE FROM THE CLINIC

OF

MR. W. H. A. JACOBSON, M.Ch.

EPITHELIOMA OF THE TONGUE.

THIS case illustrates the following important points: the existence of a pre-cancerous stage of epithelioma, some of the most common ways in which epithelioma is developed, its treatment and prognosis. The development of the pre-cancerous stage is almost invariably a matter of years. This man had syphilis thirty years ago, he has been a great smoker, a smoker too of coarse black tobacco, and has smoked as long as the pain would let him. He came to me nine months ago with a small ulcer on the tip of the tongue. This ulcer appeared slightly indurated at the base, and he allowed that the tongue had been sore for years. He was advised to have part of the tongue removed, but refused. The condition since has been getting steadily worse, and the patient reappeared on April 2nd, with the following conditions. None of the anterior two-thirds of the tongue could be described as healthy. It showed the following evidence of old glossitis: its healthy, smooth, delicate covering in a great measure gone, here and there replaced by bald patches, at other places heaped up into leucomata of varying density, elsewhere scarred also by old healed ulcers. On the left side of the tongue, involving the anterior third and dorsum and side as far back as the left premolar tooth, there is a ragged ulcer with everted edges and a base of uneven depth indurated like the edges.

In testing for induration, it is best to use both hands and take if possible a correspondingly healthy spot. This is a good rule in testing for induration of epithelioma, or of a suspicious specific sore on the genitals. The tongue in this case can be freely protruded, and no indurated glands can be felt. The patient is now willing to submit to an operation, on account of the persistent pain. The

clinical points to be noted are that the leucomata, the long-standing ulcer, and the baldness are all proofs of the existence of a pre-cancerous stage. The meaning of this term, a pre-cancerous stage (a term introduced by Mr. Hutchinson), is a condition or stage the duration of which is uncertain, and varies extremely, one in which inflammatory changes only are present, that is to say, alteration of epithelium and ulceration, none of this amounting as yet to epithelioma, though on the pre-cancerous stage epithelioma inevitably intervenes. Thus, if you had taken sections of the bald part of the tongue, of the leucomata or of the ulcer nine months ago, there would have been no in-dipping of the epithelial processes reaching a level of danger, and probably no "bird's nest" cells. The origin of the ulcer was probably in some bald patch or leucoma, such as is seen on the rest of the tongue. The leucoplasia becomes warty, then epitheliomatous; the bald patch, which is simply a thin layer of scar tissue replacing healthy mucous membrane, becoming fretted like a scar elsewhere and breaking down. In any case the boundary line between this pre-cancerous stage is extremely narrow. Its duration, though apparently of some months in this case, may be, according to the irritation it is subjected to, extremely brief. The following aids are useful in recognising this stage:—

The duration of the ulcer.—This man we know has had it nine months, but admits having suffered from a sore tongue previously.

Obstinacy to treatment.—It is shown by its resisting the treatment nine months ago when removal was then advised.

The age of the patient is useful—anything over forty or forty-five when the tissues are likely to degenerate. Habits which tend to cultivate the affection, such as smoking strong tobacco, and drinking "strong waters;" lastly, the absence of any induration or fixity. The fact that induration is present now, when tested in the way I have just mentioned, shows that it is no longer in the pre-cancerous stage. As regards the importance of this pre-cancerous stage, it may be difficult for you to realise it from this comparatively small ulcer, but it is beyond doubt that of all the painful deaths by which men are called upon to leave this world there are few more miserable or distressing than cancer on the tongue. The

patient such as this one before us with this small and apparently unimportant ulcer just beginning to call his attention to the pain preventing his smoking, gives very little idea to you of the later stages,—the hideous sore, converting the mouth and sometimes the side of the neck into vast chasms, the horrible foetor, the profuse and foul salivation; the pitiless, incessant wearing and racking aching of the tongue (or of the remains of it), the pain, deep boring into the ear, always present, and lit into flashes by attempting to take food or to speak; the dribbling of foul stinking saliva, half poisoning the patient, and rendering him noisome to others. This is, of course, in the latter stages of the condition that you see before you now. The case also illustrates the way in which cancer can be "cultivated," to use another expression of Mr. Hutchinson's. In a better rank, the patient's friends would ask you when you advise early operation, "Is this really cancer? Can you swear to it?" They expect you to give a prompt and decided answer. If this question be answered without decision they probably decide to give drugs, as they call it, "another chance." As is the case with this man, there is almost certain to be a history of syphilis; because iodide of potassium has not done much good, mercury is tried, or because the potash salt has failed the ammonium or soda salts may be substituted, or worse than this, caustics may be used. Always bear in mind in these cases that iodide of potassium, sometimes in malignant disease, in ulceration of the larynx and so on, may produce temporary benefit by diminishing vascularity and inflammation as it does about a gumma, and a patient feeling slight benefit for the time may lead the medical man into losing still further precious time. As regards caustics, the careful and thorough application of acid nitrate of mercury is sometimes justifiable. It was tried nine months ago in this case, and failed to start any real healing. On the other hand, the use of such caustics as nitrate of silver, at short and frequent intervals, is futile, highly perilous, and I might almost say that any medical man to spend time in dallying with caustics, even at the entreaty of his patient—I could almost say that any medical man thus to deal even at the entreaty of his patient with caustics or specifics, when the time has gone by—is well-nigh criminal. The dallying with drugs and local applications only cultivates the cancer, and

leads to a most miserable death. There are other questions which you may have to answer, especially in a better rank of life than this patient is in—"If I submit to the operation you propose, shall I be permanently cured?" Now in this case, where the patient has waited certainly nine months, where the induration is marked, where actual ulceration has been present many months, you will be wiser not to answer that question off-hand, even at the risk of losing the patient. It all turns on how long actual cancer or epithelioma has supervened on the pre-cancerous stage which we saw nine months ago. Given epitheliomatous ulceration, the lymphatics must be opened, and it is a mere question of time how soon epitheliomatous deposits will be taken up. Gland infection in these cases begins very soon after the time when the sore assumes *suspicious* features, features short of the induration before us. Another point of importance is the lymphatic glands may become involved with epithelioma, through an ulcer, even though of very brief duration and of most insignificant size, as long as the ulcer is epitheliomatous. Knowing this as I do, and knowing that his sore was suspicious nine months ago, my prognosis in this case would be a very guarded one, I might say an unfavorable one. Many of you have been feeling his neck, and we are all agreed that we can feel no enlarged glands; remember that in these multitudes of glands in the neck there may be deposits already taken up from that ulcer, of the minutest size, quite beyond our powers to feel. Remember, again, that these deposits may remain latent in the lymphatic glands and the neck for years, and then produce disease when the patient considers himself absolutely safe.

(To be continued.)

CLINICAL NOTES.

WITH MR. BUTLIN IN THE WARDS OF ST. BARTHOLOMEW'S HOSPITAL.

Warty Growth of the Tongue.

THIS man, æt. about 50, has a warty growth on the border of the tongue, which partially involves the mucous membrane. It is screwed down on to

the anterior half-arch, and there is a scar resulting from the removal of a portion of the tongue five and a half years ago, by Mr. Baker, presumably for epithelioma. The patient first noticed it growing five weeks ago. In this case we have practically to decide between warty growth and epithelioma; his age admits of it being papilloma.

The patient has no enlarged glands; the growth is not hard, nor indurated about the base, therefore the probability is that it is not infiltrating. The surface is not ulcerated, and there is a fur on the tongue, probably due to organisms. There are one or two tiny leucomata on the mucous membrane of the tongue, so I presume that at the time of the first operation he had chronic superficial glossitis, from which the outgrowth originated. I do not regard it as secondary to the epithelioma which was removed five and a half years ago, but as a separate outbreak of disease upon a surface which is not quite healthy. People with chronic glossitis are very liable to these warty growths; and that is the way in which epithelioma usually begins, more frequently in that way than in an ulcer or a crack or fissure; therefore, when a patient who is the subject of old-standing tongue disease comes with warty growth, the correct course is to remove it, by taking away a part of the tongue. (The growth proved, on examination after removal, to be a compound papilloma.)

As to prognosis, the operation is not very dangerous, and may be regarded as a comparatively slight one. The forecast as to the subsequent course is unfavourable because the patient seems peculiarly liable to disease. Does operation cure any case in which the growth has been proved to be epithelioma by microscopical examination? Yes, sometimes. I know some surgeons hold an opinion contrary to this, but I operated nine years ago on two men for cancer of the tongue; one of them is now between 80 and 90 years of age, and neither of them has had any recurrence.

Large fungating Melanotic Sarcoma on a Male Breast.

This man is æt. 40. Eighteen months ago, he noticed that the skin on the left breast was blue in colour, and it itched very much; then it ulcerated, and "broke out" while he was taking a dive. When a patient comes to you with a tumour in the breast, do not be content with an examination of

the breast merely ; inspect both breasts and both axillæ. If you omit this, you may get into trouble through operating on a patient who ought never to have been touched. Enlarged glands can be felt above the clavicle on both sides, and in both axillæ, though on the left they are larger than on the right. The growth may be either a melanotic sarcoma, or an ulcerated carcinoma of the mammary gland. Against the probability that it is carcinoma is the sex of our patient. Sir James Paget stated that carcinoma of the breast in men amounted to about 2 per cent. of the total cases. Gross collected a hundred and two cases, and two of them were in males. In males, carcinoma of the breast may assume the scirrhouss form ; indeed, you may even find the actual withering scirrhus. But this growth is markedly fungoid, and by passing a probe around it one is able to make out a comparatively narrow pedicle. Moreover, it moves very freely with the skin, and does not appear to have any deep attachment. Another point is that it would be unusual for the glands on both sides to be enlarged in carcinoma. What is there about this case which makes epithelioma unlikely ? First, the rapidity of the growth ; second, the fact that he has an enlarged gland on the opposite side to the growth. Lastly, what feature in this case is against melanotic sarcoma ? It is a very unusual disease, and one is naturally suspicious about people having unusual diseases ; if it be a question between two or three diseases, prejudice is always against the rarer disease, and in favour of the commoner. Now let us see what there is in favour of its being melanotic sarcoma. The fungating surface, the absence of induration, the fact that it is not deep-seated, all increase the likelihood that it is such a growth, and the fact that both axillæ are affected does not detract from the view. About the umbilicus may be seen small blue, hard masses, which we are told have only been noticed during the last day or two ; any idea that they might be gouty or accidental nodules would be disposed of by their colour, and we are forced to the conclusion that they are of similar character to this more conspicuous growth, viz. melanotic sarcomata.

Melanotic sarcoma is very rare indeed ; we do not have more than one case of it in this hospital in a year. It is not uncommon in young people the last case I had was æt. 22.

It usually begins in a blue spot or wart, which may have existed during any length of time, even since birth. Indeed, it is not uncommon for the seat of the trouble to be a pigmented wart which has existed from birth, and has remained unaltered until thirty or forty years have elapsed, when it may begin to grow, and sometimes ulcerate. It may contain either round or spindle cells, and the pigment is sometimes contained in the meshes between the cells, and at other times in the interior of the cells. If the eye be removed for melanotic sarcoma, the most common seat of the secondary growth is the orbit ; after that, many organs and tissues of the body may be implicated. If the skin of the chest be affected, nodules may occur in the skin and subcutaneous tissue, even sixty or seventy have occurred in the same patient. The lymphatic glands are peculiarly liable to implication, and one axilla may become involved very soon after the other.

The prognosis in such a case is absolutely fatal ; I do not think any treatment affects it at all. A young fellow came to me who had had a wart on the front of his chest. His doctor snipped it off ; it grew again, and he again snipped it off, and applied a caustic. When it grew again, it was surrounded by six or eight small nodules. Then the glands in both axillæ became enlarged, and I removed the whole of the foci, and the patient made a very excellent recovery. Then the disease recurred in spots about the skin in various parts of the body, and after eighteen months the patient died.

I saw a very curious case once, which points to the necessity for extreme care. Sarcoma may occur at the end of a finger ; when it does it is usually nodular, it fungates, and is either bluish or black. It affects the lymphatic glands very early, and is thus as fatal there as in any other part of the body. I have seen sarcoma in a toe, and the lymphatics in that case also were affected at a very early date. The limb was, I think, amputated. The case I particularly allude to was a young girl æt 15, who was brought by her brother, a medical man. She came for melanotic disease at the end of the finger, in three curious black places. These had been removed, and had recurred each time. At first I thought it was melanotic sarcoma, but the more I looked at it, the more I became doubtful in my own mind. She had no lump, no enlarge-

ment of the glands, and no ulceration. I cut it away and removed the whole of the black material, leaving a sore surface. I had sections made of it and examined them under the microscope, when I found an area of black stuff, with the epidermis around it a little thickened, but no spindle cells, or anything of the kind. Thereupon I formed the suspicion that the young lady had done it herself. When it had been carefully examined I wrote to say that it was not melanotic disease, and that I believed she did it; at the same time I advised the brother to have a consultation if he wished the finger to be amputated. No further opinion on the matter was taken, and several months afterwards he wrote saying it had recurred for a short time, and that at last the young girl had confessed that she put some ink under the epidermis. The strange part of it is that she would have submitted to amputation rather than confess that she caused the condition herself. I looked the matter up, and found, in the Pathological Society's 'Transactions,' a similar case of a girl, shown by Mr. Godlee. Her finger actually was amputated, and was given to him for examination. It presented an appearance exactly like my case. Pursuing my investigations, I found that the same girl, several years later, suffered from an obscure affection of the hip, which got well.

This poor man's growth will be removed, to relieve him from the ulceration, but it will recur.

Tubercular Abscess of Foot.

This young woman, *aet. 15*, came six weeks ago with a swelling in her foot, which had existed for six weeks. It was opened in one or two places, and she ought to have come to the surgery the next day, but did not do so for several weeks, and then her condition was much the same as you see it now. When it was opened on the first occasion, pus was evacuated, and it appeared to be an ordinary abscess of the foot. The three most likely diseases under which this could be included are (1) tubercular disease, (2) syphilitic disease, (3) sarcoma; perhaps we may add a fourth—actinomycosis. Against a diagnosis of syphilitic disease is the fact that it is very painful; and it would not be filled up in the way you see, but would have a scooped-out appearance, with the edges overhanging, and in tertiary syphilis one would be able

to find gummatous, and the whole affected area would be more nodular. Points unfavourable to sarcoma are, that the locality is not that usually chosen by such cancerous growth; only once or twice in my life have I seen such a neoplasm in the foot. Sarcoma of the lower extremity is common between the ages of 15 and 25; I have only recently discovered the fact in investigating sarcoma of the long bones. In my own mind I have attributed this to the fact that at that age the growth of the bone is not perfect, and at the same period a good deal of exercise is taken. Sarcoma is less common after 25 and before 15, while in childhood it is a very rare disease. Sarcoma would not have broken down in the way this has, nor at so early a period, and there would be no pus; moreover, sarcoma would bleed more than this has done. What can be urged against the opinion that it is tuberculous? Very little. The age of this patient is a very common one for tubercle, and the situation of this outbreak is also common. Generally, tuberculous disease affects a number of bones. The swelling is elastic in the centre, while at the outer edge one can detect bone beneath, and there may be a considerable amount of granulation tissue; in fact the bone has probably been blown out by the tuberculous disease. Or it may be due to spiculae of bone being close to the surface. The ulcer is very typical; circular, fungating, pallid granulations, the skin around being a dull livid colour, and a probe passes into soft bone. The discharge appears to be ordinary pus, and there is considerable pain; the temperature is slightly raised, but does not oscillate with regularity. Tubercle bacilli have been found in the granulations, so that our diagnosis may be considered confirmed.

The treatment consists in scraping away the bone and removing all the granulations, afterwards packing with gauze; iodoform and oil, or iodoform only, may also be injected.

The prognosis is doubtful. It is right for us to make an attempt to save the foot. Our treatment will shorten the foot, but the sore may heal up and the foot may be serviceable.

A NOTE FROM THE CLINIC
OF
Mr. GEORGE POWELL, F.R.C.S.,
At Westminster Hospital.

IRIDECTOMY.

THERE is a case in St. Matthew Ward on whom, last Tuesday week, you saw me perform the operation of iridectomy. The man, who comes from Suffolk, is very rheumatic, and some years ago had an attack of rheumatic iritis. Last year he had a recurrence of the inflammation, and quite recently has had a third attack in the same eye, which did not yield to treatment, and for which he was sent into the hospital. Under the continued use of atropine in the eye, and the administration of moderate doses of iodide of potassium, the iritis yielded, and it was found that there were numerous posterior synechiæ. The presence of these adhesions, the double recurrence of the inflammatory attack, and the considerable deterioration of vision, led me to perform the iridectomy, not by any means because such a proceeding is a routine treatment in cases of iris adhesion, but because the rheumatic origin, the double recurrence with only a short interval between the attacks, and the probable implication of the choroid, called for some such radical interference. I can, indeed, remember the time when this operation was thought necessary in all cases of iris adhesion after iritis, and especially if recurrence of the iritis occurred. I have raised my voice more than once against it, and now it is fully recognised that it is by no means certain that iris adhesion will be followed by a recurrence of the iritis. The operation is now hardly ever performed when the posterior synechiæ have been due to iritis of a syphilitic form. It is, however, more frequently required in rheumatic cases, and especially when recurrence seems established, or where the eyes show some slight increase of intraocular tension. It is found in such cases that iridectomy not only diminishes the tendency to recurrence, but tension is diminished and vision is improved. In all cases, too, where adhesion is so extensive that the whole margin of the iris is adherent, blocking all communication between the

anterior and posterior parts of the anterior chamber, iridectomy is a necessity, or the pressure of the aqueous accumulating behind the iris will cause ballooning, and give rise to a train of changes which will result in destruction of vision.

The operation of iridectomy performed in the case before us, and its already happy effect, has suggested the subject of my lecture to-day. Iridectomy, or the removal of a portion of the iris, is performed with a variety of objects which may be divided into therapeutical or physiological and optical, and the manner of making the iridectomy, the size of the portion of iris removed, and the position of the iridectomy, will vary according to the object that the surgeon has in view. Von Graefe was the first surgeon to introduce the use of iridectomy as a therapeutic means of arresting the changes in the eye which are produced in various forms of disease. He established its value in dealing with the intraocular tension which is present in various degrees in the common pathological process to which has been given the name of Glaucoma. Von Graefe has given us his experience in the use of iridectomy in the premonitory stage of glaucoma, in acute glaucoma, both early and late in the progress of the disease, and also in the more chronic forms, and in the excavation of the optic nerve which accompanies the blindness so frequently the result of this disease. There has been but little modification of his views by other surgeons. Other means of relieving tension have been devised, but all have their special dangers and shortcomings, and iridectomy as a therapeutic agent continues to hold its own. It is still held that a large iridectomy in which the portion of iris taken away is cleanly removed from its peripheral attachment is the only trustworthy cure for acute Glaucoma. In the other forms, the iridectomy is not required to be so large, and in fact my own view is in favour of diminishing the size of the iridectomy in proportion as the disease is chronic. With reference to the position of the iridectomy, von Graefe preferred to make it laterally. The majority of modern surgeons now prefer the upward position, in order that the coloboma may be well covered by the upper lid. The position, however, signifies little. The points of greatest importance whenever iridectomy is adopted with a therapeutic object in such a disease as glaucoma are—

1. That the incision should be made as excentrally as possible, half a line or more outside and parallel to the external sclero-corneal junction, and thus through the internal sclero-corneal junction, in order that the iris may be removed in its whole width and cleanly from its peripheral attachment.

2. That the aqueous should be allowed to escape by the side of the knife as slowly as possible, that there may not be a too sudden relaxation of support to the retinal and choroidal vessels in the tense eyeball. The greater the tension the more important is carefulness in this matter, as in consequence of the great tendency to rupture of vessels in such cases, a sudden relaxation of pressure is sure to be followed by undue ecchymoses, and sometimes in the more acute cases even by haemorrhage, which has been known to force out the contents of the eyeball. Von Graefe tells us that so important he considered care in this respect that his practice was to exert pressure on the globe with his finger, and immediately after the operation to apply a compressing bandage, to be cautiously slackened in a few hours. As an additional point that must be mentioned, in making the incision great care must be taken to keep the point of the knife away from the lens, lest it be wounded. A too rapid escape of the aqueous may sometimes conduce to this accident. The size of the iridectomy must be proportioned, as I before stated, to the acuteness of the case. Acute glaucoma requires that one third of the iris should be removed. The less acute the attack the smaller the iridectomy, one sixth being ample in the more chronic cases.

Besides the varieties of glaucoma, there are other cases in which iridectomy is therapeutically advantageous. When the choroid is implicated either from an extension of inflammation in keratitis or iritis, or both, vision often rapidly deteriorates. Tension may be increased, and it frequently is at first, or tension may be diminished. In both conditions a moderate iridectomy produces an extraordinary amount of mitigation of symptoms, relieving tension in the one case, and arresting the progress of pathological change in both. In papillary exclusion, to which I have already alluded, iridectomy is peremptorily called for. In some severe cases of corneal ulceration, where antiseptic treatment, cautery, paracentesis, or Saemisch's

operation has failed, an iridectomy may succeed in arresting the threatened destruction.

For optical purposes iridectomy is necessary in a variety of cases. Such are opacities of the cornea, in a position covering the pupil more or less; central cataracts, either capsular or lamellar, when the peripheral part of the lens is clear; of course, occlusion of the pupil; some cases of dislocated lens; and some cases of conical cornea. The method of performing iridectomy for this very different object varies completely from that I have been describing. In this set of cases there is no therapeutic object, and therefore no necessity for removing the iris from its peripheral attachment. The incision should therefore be made at the external corneal edge or sclero-corneal junction, and only so much of the iris taken away as is absolutely necessary for the object in view. The smaller the portion taken away the better, vision being clearer through a small pupil, even in emmetropic eyes. The less peripheral the pupil, too, the better the resulting vision. With regard to the direction of the coloboma, the cases of corneal opacity often leave us little choice, and the clearest portion of cornea is a tolerably definite guide. In such cases, if there is room for any choice the position downwards and inwards is the most useful in the majority of patients, and especially in central and lamellar cataracts, when both eyes have to be similarly treated. The next best position is directly downwards.

The last set of cases for iridectomy that I shall speak of is as a part of the operation of extraction of cataract. I am very strongly in favour of this proceeding as one of the steps of this operation, although in these modern days foreign ophthalmic surgeons, or the majority of them, are extracting without it. When cataractous lenses were first extracted through the smaller incision, iridectomy was universally adopted to facilitate the escape of the lens. In addition to this facility it was a means of minimising two great dangers, viz. the prolapse of the iris through the wound, and the occurrence of secondary iritis. Perhaps it is the introduction of the use of eserine that has led to the more general attempt of avoiding the so-called mutilation of iridectomy in this operation. Although I do not consider that the upward iridectomy is a mutilation, I adopted this practice in a large number of cases; but I have gone back to the iridectomy, except in

a few picked cases where the eyes seem especially favourable for omitting it, as I found that my average of success was much greater with than without iridectomy. This is not the place to go into this question fully; but with reference to the two points that I have named, with iridectomy prolapse becomes impossible and the occurrence of iritis is exceptional; whilst without iridectomy, the effect of eserine is so evanescent that prolapse is of very frequent occurrence, and iritis is much more frequent than when iridectomy has been performed. This latter fact was often observed by the old operators, who noticed how well eyes recovered when an accidental iridectomy was made during the old flap extraction. If we remember that neither prolapse of the iris nor iritis can occur without interfering with the healing of the wound, and postponing convalescence to a more or less dangerous extent, it is at once explained why complete recovery of vision should be rare when either of these untoward events occurs. A round mobile pupil is no doubt a great *desideratum*, but it is so often neither round nor mobile that I prefer a good general average of good vision in my operations to a few typically perfect results, and a majority of cases in which the results are more or less imperfect.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, North-West London Hospital,
May 20th, 1896.

DR. LONG in the Chair.

Two Tumours in the Head of an Infant.

Mr. JACKSON CLARKE showed a young baby with two tumours on the left side of its head. The child was brought to him in the out-patient department, and its functions appeared quite normal. The tumours were situated over the upper part of the squamous suture. One measured $1 \times \frac{3}{4}$ in., the other $\frac{3}{4} \times \frac{1}{2}$ in. Three possibilities arose in cases of congenital tumours of the scalp, viz. meningocele, encephalocele, and dermoid tumour.

Dermoids were generally single and subcutaneous, although often connected with the dura mater. It was necessary to remember that the brain was merely an epidermic structure, and that some dermoids were quite shut in by the skull and dura mater, lying occasionally in the substance of the brain. Meningocele ought to be more or less completely reducible within the cranial cavity; but in the child before them that was not the case. Moreover, meningocele, being fluid, should fluctuate, and should become tenser when the child cried. This child's tumours were firm, and the skull appeared deficient at their bases, while a piece of cartilage or bone covered the convexity of the tumour. The only fluctuating spot was at the back part of the larger and anterior tumour.

The tumours did not pulsate, therefore he had concluded that something had formed under the skull and elevated up what could be called normal Wormian bones, and there was a gap in the skull where that bone would have fitted in had it not been so elevated. Probably the source of the displacement was the growth of a meningocele, but the two other kinds of tumour he had mentioned could not be excluded, because the Wormian bone would mask any pulsation. He would like the opinions of members on the case.

Dr. CLAYTON asked whether the labour was instrumental, and whether Mr. Clarke had considered the possibility of the tumours being haemorrhagic. The position was not a usual one for cephalo-haematoma, but the rim of bone perceptible in the case was similar to that connected with the latter condition.

Mr. JACKSON CLARKE, in reply, said the labour was a natural one. Cephalo-haematoma in a new-born baby was generally subperiosteal, and followed the shape of one of the bones, stopping at the sutures. In the child exhibited, not only was a margin felt in the skull, which might be simulated by haematoma, but there was a definite cap of bone to the swelling.

The CHAIRMAN asked what treatment Mr. Clarke proposed.

Mr. JACKSON CLARKE said he would watch the progress of the case, instructing the mother to prevent the child receiving knocks or bumps on the head. If his view was correct, he hoped the Wormian bone would spread out as the child grew, and the gap thus become sealed up by bone.

Case of Recovery after Removal of Loose Body from Knee.

Dr. LEWIS showed, for Mr. Durham, the young man from whose knee-joint the latter gentleman removed, on February 17th, a loose body ("CLINICAL JOURNAL," p. 375). There was almost perfect movement of the joint in every direction.

Mr. JACKSON CLARKE, after reminding members of the case, said an old but now obsolete method of treatment of these loose bodies was to make a subcutaneous incision into the synovial membrane, and force the body into the subcutaneous tissues; the body was not removed altogether. Antisepsis, however, disposed of much of the risk of opening the joint, and it was much better to get rid of the substance completely.

Two Cases of Aneurism.

Dr. HARRY CAMPBELL showed two men the subjects of aneurism. The first man, who had an abdominal aneurism, had been under his observation more than two years. Two years ago he was nine months in the hospital, previous to which he was a saddler in the army, subsequently following the occupation of horse-slaughterer. He had the unmistakable expansile tumour in the abdomen, and was subjected to the ordinary treatment in the hospital, but without much alteration. Since leaving the hospital he had followed his ordinary occupation. There was a history of specific disease. One part of treatment which he had carried out consisted in placing the patient under chloroform, and applying a tourniquet to the abdominal aorta, high up. The swelling diminished for the time, but afterwards returned to its original dimensions. The question arose, how far should a patient with aneurism be kept at absolute rest? Dr. Goodhart, he believed, did not keep such cases in bed, but allowed them to walk about. The patient before them kept at work every day, was frequently galloping on horseback, and did not seem to greatly respect the counsel to keep quiet. He, Dr. Campbell, would like the opinion of the Society as to the particular vessel affected. The patient was not taking medicine.

The second patient, who had aneurism of the arch of the aorta, was æt. 43, and here also there was a distinct history of specific disease. When he entered the hospital there was a very marked

thrill at the right base, but no musical murmur, and the dulness increased to the right of the sternum. The man's occupation was that of plate-layer. He would like to know whether members of the Society would condemn such cases to perpetual rest, or allow the ordinary avocations to be followed.

Mr. JACKSON CLARKE, speaking on the diagnosis of abdominal aneurism, said the closest simulation of this condition was sometimes observed when a small solid tumour lay in front of the abdominal aorta. He had known a tumour of the great omentum to be mistaken for aneurism, but when the patient was put on his hands and knees the solid tumour could be grasped quite free from the aorta, and was thus found not to pulsate. The history of syphilis in both cases was important. Sir William Broadbent had instilled into his mind the fact that a considerable proportion of cases of aneurism were attributable to syphilis, and in the course of making a great number of post-mortem examinations that lesson had been fully confirmed. The heart itself was also often affected by syphilis. In a case of sudden death which he had investigated that day there was a large fibrous patch in the wall of the heart. A tertiary lesion had gone on for several years, and had at length caused syncope. Where there was any doubt he strongly advised the administration of iodide of potassium.

Dr. CAMPBELL said that, although he had never had a case of syphilis of the heart, it was wise to bear the possibility of such cases in mind. Quite recently, at the Clinical Society of London, Sir Dyce Duckworth brought forward a case of gumma of the heart which had caused sudden death. One member present, after searching the records, was able to allude to six or seven other cases of a similar nature.

Tertiary Syphilis.

Mr. JACKSON CLARKE showed a woman who came to him to be treated for baldness. Cursory inspection led him to think it was alopecia areata. The patient was weak and anaemic, and, curiously, there was no reddening of the part. He used the usual stimulative treatment for alopecia areata—iron internally and sulphur ointment locally. Next time she came he was puzzled by her anaemic appearance, and inquiry elicited the usual history of

miscarriages. On the centre of the tongue was a little nodule of recent formation, which no doubt was a minute gumma. This and two other lesions, which appeared subsequently on the tongue, cleared up under iodide, and the patient's general health had greatly improved under the treatment. The case shows that syphilis can never be searched for too often. The rapidity with which hair came back on five-grain doses of iodide of potassium was very remarkable, while the pallid aspect of the patient had changed to a look of health.

Dr. Campbell remarked on the absence of headache in Mr. Clarke's case of tertiary syphilis. His experience was that headache tended to accompany the extension of syphilis to any part of the cranium. Neuralgic pains in the head were frequently associated with alopecia areata. A great German authority on syphilis pointed out that attacks of cerebral syphilis, as he called them, were very prone to be heralded by extreme headache. In one half the cases of syphilitic paraplegia, as Dr. Gowers had pointed out, there is severe headache just before the stroke, and it was thought that if they carefully looked for them they would, in those cases, find extra-cranial syphilitic lesions.

Dr. CLAYTON, referring to the case of syphilis, asked whether in tertiary syphilis the alopecia was not, as a rule, more diffuse than described by Mr. Clarke. He would also like to know whether the stumpy marginal hairs, which had been likened to a note of exclamation, and which were usually regarded as diagnostic of alopecia areata, were present.

Mr. JACKSON CLARKE said he confessed he did not particularly look for the hairs mentioned, but there was a sharply defined patch of areata, not the diffuse form. Regarding Dr. Campbell's observations, the patient did not complain of headache, but she had giddiness, which might be accounted for by the anaemia which was present. He had had syphilitic patients under him for three or four years, who did not complain of headache until the end of that time. One patient had had all sorts of lesions, in the throat, skin, lymphatic glands, and bones, and was at the present time complaining of headache, though the dose of iodide she was taking was only moderate. The patient might possibly have some meningeal

thickening or even some thickening of the frontal bones, but she continued to be cheerful.

Tumour of the Parotid.

Mr. JACKSON CLARKE exhibited, for Mr. Mayo Collier, a young woman with a large protuberance under the ear, a condition which she had had ten years, but it had caused her no pain. The tumour was slightly lobular, and felt as if mostly made up of cartilage. Sections of such growths revealed not only cartilage, but also structures which looked like gland-acini cut across. Such growths could be classified as chondro-adenomata, which might pass on to chondro-sarcomata, taking on a rapidly malignant course, though the diagnosis between malignancy and innocence was chiefly a clinical matter. These tumours in some cases were not difficult to remove; but in others part of the tumour might be found dipping under the temporo-maxillary vein and the external carotid artery. Even then the tumour was so definitely encapsulated that it could be removed, usually with some damage to the facial nerve. When facial paralysis followed such damage it usually passed away.

REVIEW.

Diseases of Rectum and Anus, and Contiguous Textures. By S. G. GANT. (F. A. Davis & Co.) Price \$3.50.

Truly are the Americans an original people, when with a book a review is sent for the reviewer's guidance. We must admit, however, that the points given are true in substance and in fact, and the book is a good one for students and busy practitioners. The plates are excellent types of what coloured plates should be; and the woodcuts, though mostly diagrammatic, are clear and distinctly useful. That a book written in American style, with American spelling, can ever become a real favourite with English readers is hard to believe, but the specimen under consideration will, we think, from its subject arrangement, have a very good chance of doing so. We can certainly warmly recommend it to those whose time is limited, who yet wish to have some special information on some common but distressing diseases. It is, however, mainly written for surgeons by a surgeon.

THE CLINICAL JOURNAL.

WEDNESDAY, JUNE 10, 1896.

A NOTE FROM THE CLINIC
Of BILTON POLLARD, B.S., F.R.C.S.,
AT
The North-Eastern Hospital for Children.

Ectopia Testis.

THIS is a case of a well-known but rather unusual malformation. It is a child æt. 3 months, and you notice the right side of the scrotum is unusually small, and the raphe is not in the middle line. No testicle can be felt on the right side of the scrotum, but you see there is a swelling in the perineum a little in front and to the right of the anus; that swelling contains a firm substance resembling a testicle, and from it can be traced upwards to the external abdominal ring a band which resembles the cord. Though this is an unfrequent malformation, I have had perhaps unusual experience of it, in that already I have operated on two cases very similar to this, though in those the testicle was a little further forward in the fold between the scrotum and the thigh, and at the present time I have another case under my care at University College Hospital which I shall operate on shortly. I have also had a still more unusual and almost unique example of ectopia testis; in that case the testicle lay on the dorsum of the penis close behind the corona. These malpositions of the testis are very interesting in relation to the mode of descent of the testicle, which depends, as you know, on the action of the gubernaculum testis. This muscle is attached to the bottom of the scrotum, to the pubes, to the root of the penis, in the perineum in the neighbourhood of the anus and tuber ischii, and to the fascia over the front of the thigh. Ordinarily the fibres attached to the scrotum prevail and cause the testis to assume its proper position, but in some cases, of which our patient is an example, the other fibres succeed in dragging the testicle to one or other of the sites mentioned. In the three cases that I have operated upon the tunica vaginalis

surrounded the testicle in a normal manner. This child will be operated upon in a few days, and the method which I adopt, and which is slightly different from that recorded by other surgeons, is as follows:—I make an incision extending from the external abdominal ring along the cord to the testicle; I strip the testicle in its covering from its bed, and turn it upwards to the external abdominal ring. I then make an incision into the lower part of the empty half of the scrotum, of sufficient size to accommodate the testicle. I then pass a pair of sinus forceps from this incision up the scrotum to the upper end of my first incision, in the neighbourhood of the external abdominal ring, and separate the blades sufficiently to make a track along which the testicle can with some pressure and manipulation be forced. The testicle is now shoved along this track to the bed prepared for it at the bottom of the scrotum. The reason for proceeding in this way is to prevent subsequent displacement of the testicle, for the track along which the testicle has been passed at once shrinks round the cord, and prevents the testicle being retracted. The wound is closed and dressed in the ordinary way. Some surgeons have advised delay until the child is two or three years old, in order to facilitate antiseptic surgery; but as in cases of radical cure of hernia, when essential, I have found no difficulty when operating at quite an early age. In one of my previous cases the child was only two months old when I operated; of course, if the malposition has been unrecognised or neglected until a later period, it is still very desirable to replace the organ in the scrotum. The transplantation of the testicle from these malpositions is generally quite easy, and in marked contrast to cases of retention of the testicle in the groin, or in the upper part of the scrotum. In cases of ectopia testis that I have seen the spermatic cord is amply long enough to allow the testicle to be placed at the bottom of the scrotum. In cases of incompletely descended testicle the cord is short, and upon this depends the great difficulty in placing the testicle in its proper site. Mr. Watson Cheyne has devised a wire cage which fits into the

perineum over the scrotum, with a cross-bar to which a silk thread attached to the lower part of the tunica vaginalis and brought through the bottom of the scrotum, can be tied. This is, I believe, the best means of holding the testicle down whilst the wound is healing, but even this plan in my hands has not succeeded in preventing a shortened cord from drawing the testicle somewhat higher up than it had been placed at the operation. I have used the cage in one case of ectopia perinealis testis ; it acted very well, but I came to the conclusion that, owing to the length of the cord, it was not really required.

Acute Necrosis and Epiphysitis contrasted with Tubercular Epiphysitis.

This boy, aged five years and nine months, is, as you see, very ill. He was admitted to the hospital on the 13th of April, 1896 ; he had kept his left leg drawn up, and complained of great pain about the knee for three days previously to admission. He has been very feverish and has lost all appetite. There was no history of injury. On admission the boy was found to be well nourished, though pale, fretful, and obviously very ill : his temperature was 102° . His left leg was flexed at the hip and knee, and a little above the latter joint the thigh was swollen, hot, and acutely tender; no fluctuation could be detected. The knee-joint appeared to be unimplicated. An incision was made on the outer side of the thigh, a little above the level of the upper border of the patella ; this was carried down to the bone, and about half a drachm of pus was evacuated from beneath the periosteum, the bone was denuded down to about the level of the epiphysis, the cavity was freely irrigated, and a drainage-tube was inserted, antiseptic dressings and a splint with a foot-piece were applied. On the following day the boy's condition was improved, the temperature was lower ; the wound was again irrigated and redressed. On the 15th of April the boy was not so well, he was rather delirious, and his temperature was 102.5° . An incision was made in the inner side of the thigh, opposite the original incision, and carried down to the bone, and the cavity around the femur, which was found to have increased in extent, was thoroughly flushed out with 1 in 2000 solution of perchloride of mercury. The knee-joint was now found to be swollen and to contain fluid; some of

this was drawn off with an aspirator, and it was found to be serous. On the afternoon of the same day the wound on the outer side of the thigh was enlarged, and another collection of pus behind the femur was evacuated ; after this the temperature fell gradually during the next few days to 100° , and the patient's condition again improved. On the 18th, the knee-joint having become more distended with fluid, it was again explored and pus was evacuated. Free incisions were then made into the joint on either side, and the cavity was washed out and drainage-tubes were inserted ; the temperature now fell lower, reaching the normal level in the morning, but ascending to between 100° and 101° in the evening. The boy was still very fretful, his tongue was very furred, he was occasionally sick and had diarrhoea ; there was no evidence of pus being pent up, but his condition still remained unsatisfactory ; so on the 9th of May the incision into the knee-joint on the outer side was enlarged, and the cavity was explored with the finger. At the upper border of the trochlear surface of the femur a bare piece of bone and a sinus into which a probe could be passed were found. This sinus corresponded with the position of the epiphyseal line, the sinus into the bone was enlarged, and a considerable quantity of necrosed suppurating tissue was scraped away.

As the drainage from the joint did not seem to be quite perfect, a counter-opening was made in the popliteal space and a tube inserted. Since then the dressings were changed twice a day, and the joint and abscess cavity round the femur irrigated out. The boy is now improved, his temperature keeps lower, and the drainage is satisfactory. As a matter of fact, the tube inserted through the popliteal space is not doing much good ; it gets nipped between the bones, and this is in agreement with my previous experience in regard to this method of draining the knee. The case is chiefly interesting through the implication of the knee-joint. The explanation of this is that the disease either began in or subsequently extended to the epiphyseal line, and had burst through the portion of that line which corresponds with the knee-joint ; this is the usual cause of suppuration of the knee-joint in cases of acute necrosis of the lower end of the femur, and a frequent cause of acute arthritis in infants. In some cases of acute necrosis, simple serous

synovitis occurs. Suppuration does not occur, and the fluid is absorbed. It is, however, very important, if there is fluid into the joint and the temperature keeps up, to aspirate the knee in order to determine the nature of the fluid, with a view to adopting suitable treatment if it be purulent. In cases of infective inflammation of the lower epiphyseal line of the femur in infants the disease does not always extend to the joint, but in such cases as I have seen in which it has done so there has been a sinus passing into the bone at the same spot as there was in this case. A considerable portion of the epiphyseal line does not abut upon the joint, and so it is possible for the pus to escape from the surface of the bone either on the inner or outer side or behind, and so the joint is not affected. At the upper epiphyseal line of the femur the position is different; there the epiphysis of the head of the bone is entirely articular, and so suppurative epiphysitis nearly always leads to disease of the joint,—indeed, this accounts for a certain number of cases of hip-joint disease which progress very satisfactorily after operation; they are not tuberculous, and there is not the same tendency to relapse after the joint has been thoroughly cleared out and the diseased bone removed.

In contrast to the above case I show you this little boy of three years of age. His mother noticed a lump on the inner side of the right knee about six weeks ago; it was painless and was disregarded, the child was able to walk and to use the knee-joint freely. About four weeks ago it was brought to the hospital, and a swelling was found over the inner condyle of the femur. It was not painful, and no fluctuation could be detected. The joint was fixed by means of a splint, and cod-liver oil and steel wine were ordered. It was thought to be tubercular disease involving the lower epiphyseal line of the femur. The child was not seen again until a fortnight ago, when there was an obvious fluctuating swelling situated over and rather above the inner condyle of the femur just about the junction of the epiphysis and diaphysis. It was the size of a bantam's egg, the skin over it was quite healthy, the swelling was painless, and the movements of the knee-joints were not restricted; the child was well nourished, and of a ruddy complexion.

Notwithstanding the limited nature of the disease

and the freedom of the joint, the condition was looked upon as one of considerable urgency; it was thought that tubercular disease of the epiphyseal line had extended into the surface of the femur, and given rise to the abscess which was observed. It was, moreover, thought that the disease might extend into the epiphysis itself, and possibly into the joint, unless its spread could be arrested by an operation; so three days later an incision was made over the swelling, and about an ounce of thin tubercular pus was evacuated. After cleaning out the abscess cavity a small sinus was found passing into the femur; this would only admit a fine probe. It was enlarged with a 'Volkman's' spoon, and a great deal of caseated tuberculous tissue was scraped away from the condyle. A finger was inserted, and it was found that the condyle was hollowed out by tuberculous disease. In scraping out the cavity a piece of cartilage covered with synovial membrane was removed; this showed that the disease had reached the articular cartilage and was on the point of implicating the joint, if it had not already done so to a slight extent. The cavity was irrigated, dried, and its walls were smeared over with iodoform, sterilised by boiling; the wound was closed by sutures, dressed, and a splint applied. Since the operation the temperature has remained normal, the boy eats and sleeps well, and is free from pain. This case contrasts sharply with the other in the insidious and painless onset, and progress, as opposed to the fulminating onset of the first case. The necessity for prompt treatment in the first case would be apparent to all, but it is none the less urgently needed in the second case. In the first case without treatment there would have been grave danger to life. Though this was not the case in the second, there was, nevertheless, grave danger to the joint. If a little longer delay had occurred in the latter case, there can be no doubt that the joint would have been involved in a tuberculous synovitis; as it is, there is every reason to hope that with the removal of the tuberculous focus from the condyle of the femur the joint itself will escape.

Umbilical Hernia.

Umbilical hernia is very common in children, and certainly one large factor in its production is the flatulent distension of the intestines to which ricketsy

children are liable, and in the treatment of such it is not sufficient to put on the regulation pad and bandage, but the state of the intestines should be also remedied. The case, however, which I am now showing you is not one of the ordinary reduceable umbilical hernia. In this case there was a cystic swelling distending the umbilical cicatrix; it was irreducible. The baby was said to have had the swelling of the navel soon after birth; this had disappeared, but latterly the present lump had been noticed,—chiefly, probably, owing to its being painful. The umbilicus was found to be occupied by a small apparently fluctuating swelling about the size of a broad bean; it was irreducible, and its deep connections were not ascertainable. An incision was made over the swelling and the latter was opened, a little fluid escaped, and a small mass of granular omentum unravelled itself. It was an omental hernia; the opening into the abdomen would barely admit a probe, and the omentum could not be reduced until the opening was enlarged. As the omentum was a perfectly healthy, flimsy membrane, it was thought better to reduce it than to remove it. This was done, the opening was closed by two silk stitches, the skin wound was then sutured and dressed; the temperature remained normal, and the child left the hospital with the umbilicus healed on the twenty-fifth day after the operation. It is very rarely necessary to operate upon umbilical herniæ in babies; this is only the second case in which I have operated. The other case was a still more unusual one, in that it was a case of strangulated umbilical hernia occurring in a baby of two or three weeks old. It was under my care at the University College Hospital, and made a complete recovery after the ordinary operation. The indications for operation in these two cases were in the one an irreducible hernia, and therefore one incapable of treatment by a pad and bandage, and in the other case the presence of strangulation with its ordinary symptoms.

Lumbago.—

B. Sodium salicylate	3ss.
Potassium iodide	3ij.
Compound syrup of sarsaparilla	3iss.
Water	q.s. ad 3ij.
M.S. A teaspoonful in water thrice daily, after meals.	

SOLIS-COHEN.

CLINICAL NOTES.

WITH DR. TOOTH AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC, QUEEN SQUARE.

Infantile Hemiplegia.

THIS child of three years of age is brought here for weakness of the left side of the body. The mother has noticed for the last three or four weeks that the left arm has been particularly weak. There is no history of fits or convulsions during teething. The mother says that the labour with the child was a long labour, but that no instruments were used. In walking there is a tendency for the left leg to drag. It has been taken to the Great Ormond Street Hospital for marasmus. There is more absence of history in this case than there generally is in these cases of infantile hemiplegia. One might call this a case of infantile paresis rather than paralysis. These cases generally date from some attack of fits at the teething period, but there is a complete absence of any history of fits in this case. The child is undersized for his age, but he is good tempered and very quick and intelligent, and speaks quite plainly. In the upper extremities the movements and grasp are stronger on the right than on the left. The mother says the child is very clumsy with his left hand: on the child taking up an object from this table you notice for yourselves that the clumsiness is very apparent. Though there is a slight dragging of his left foot I consider that he walks fairly well, but there is some turning in of the toes on the left side; the knee-jerks are fairly well marked, and there is not any difference between them. I think that there is a slight flexor rigidity of the left leg. The mother must think herself fortunate that the child is no worse; many of the children with this complaint are cripples for life, and I think he will be clumsy all his life on that side at any rate; but I think that he may improve considerably. Still, though it is impossible to be exact as regards the prognosis, one good sign is the fact that the clumsiness has been getting perhaps a trifle less lately. Sometimes infantile paralysis takes a hemiplegic form, and then it may at first sight be difficult to distinguish from infantile

hemiplegia. But this difficulty ceases if you consider the features of the paralysis of the spinal type, in which you find atrophy of groups of muscles with contraction of the wasted muscles and consequent deformities, and also loss of knee-jerks when the thigh muscles are affected. In this condition, on the other hand, the knee-jerks are unaltered or generally increased. There is not enough contracture in this case in the left leg for anything to be done surgically. I shall order him some cod-liver oil and malt and syrup of phosphate of iron, and we will see the child again in a month's time. The leg should be well rubbed with a little olive oil as a lubricant.

Hemiplegia.

This man, *aet. 47*, is a brewer's assistant, and is brought here for paralysis on the left side. Up to the 26th of November last he was quite well doing his work, when he had a fit, becoming quite unconscious and remaining so all night. The tongue was sore, indicating that it was an epileptoid fit. This patient is said never to have been "right" since that fit. After the fit the right arm and leg were paralysed slightly, but he was able to go on with his work. About twelve days ago a more complete loss of power in his right arm and leg supervened, coming on quite gradually, the paralysis becoming complete in twenty-four hours. The point of importance here is the gradual onset. The first attack was quite sudden, probably associated with the fit; and the second attack was not associated with the fit, and was of gradual onset. Since November the patient has had much pain in the head, and the nurse says he has become wandering in his talk at nights. The speech has been affected since the last attack; sometimes he uses words wrongly. There is a history of a considerable amount of drinking, probably in connection with his business. The patient complains of no pain in the limbs, the tongue is protruded straight, and on showing his teeth a little weakness is apparent on the right side, but it is not very marked. The pupils are now equal and react to light and accommodation, and there is no nystagmus; there is independent action of the orbicularis palpebrarum. The grip on the left side is 85, and on the right side there is no power at all; he cannot stand, and is unable to move the fingers of the right hand; the knee-jerk on the right side is

very lively, and there is no rigidity as yet to be made out. The first hint of descending degeneration is increased knee-jerks, which this patient has. What I have been trying to elicit is whether it is alcoholic neuritis of a hemiplegic type, but this increased knee-jerk on the right side seems to negative that conclusion. There is no history of rheumatism or endocarditis, and his eyesight is good. It is a small point worth remembering in hemiplegia of capsular origin that the lower face muscles are affected, but the only evidence that the action of the upper face muscles is really interfered with is that the patient cannot close the eye on the paralysed side independently. Broadbent pointed out that the upper face muscles are unaffected in hemiplegia, not because the fibres from their centres escape, but because the opposite centre, being so in the habit of working with its fellow on the opposite side, performs double work; whereas the lower face muscles are much more independent in their action, and they usually show some sign of paralysis even in the mildest form of hemiplegia. The only evidence that we can find that the upper face muscles are affected is in loss of independent action. This has been called by Revilliod, "*le signe de l'orbiculaire.*"

There is loss of power on the right side, and his grip is 85 on the left side. That leads us to remark that in most cases of hemiplegia the weakness is not found entirely on the paralysed side; nearly all cases of hemiplegia are not only paralysed on the opposite side, but they are also weak on the same side. I should judge from this that his grip, though still powerful on the left side, is not what it should be, as he presents the physique of a powerful man. One might almost say dogmatically that there is not any such thing as hemiplegia of cerebral origin; there is always a certain amount of diplegia. An important point in this case, from the view of prognosis, is that at present there is no sign of rigidity, so that the descending degeneration has not yet begun; that is, I think, a good point in this diagnosis. At this early stage I do not think we should take too much notice of the knee-jerks being increased, but they may serve as hints of possible further degenerative changes. In testing for sensation he localises fairly accurately, so I think we can exonerate the posterior part of the capsule from having much to do with the lesion. There is no cardiac murmur and no

accentuation of the aortic second sound. As a matter of interest one would like to know what the lesion is, but it is hard to say. Where there is no evidence of tension, where the apex-beat is normal and the aortic second sound is not accentuated, one would perhaps think rather of thrombosis than haemorrhage. It has been thought, in England at any rate, that thrombosis is the more common accident of the two. Dr. Nothnagel makes the statement, based probably on conjecture rather than on post-mortem records, that haemorrhage is more frequent than thrombosis. Speaking from the point of view of a demonstrator of morbid anatomy at a large general hospital, I should say haemorrhage is the commoner; but speaking as a physician to this hospital, where one sees so many cases of hemiplegia in patients, I am inclined to agree with Dr. Gowers that the most common cause of hemiplegia is vascular occlusion. All one can say is that the diagnosis between the two is at present somewhat conjectural. There is only one form of hemiplegia in which you can assert with more or less certainty what the lesion actually is, and that is in embolic hemiplegia. The attack in those cases is quite sudden, the patient is struck down quite suddenly, which he rarely is in haemorrhage, and perhaps never in thrombosis.

Functional Paralysis.

This is a case of a girl *aet. 21*, who comes complaining of loss of use of the right hand for sixteen months. The first thing she noticed was pains in the arms for several days; the pains were very severe, they were shooting in character, reaching down the arm into both sides of the hand and along both sides of the arm, so that one cannot say that it was due to any one nerve more than to another. Three days after the onset of pain the arm became paralysed, and the pain ceased. The arm has remained the same ever since; sensation is now completely lost in the hand. At first, she tells us, there was complete loss of sensation in the whole of the right arm, but the sensation recovered to above the wrist after using the battery. There was no wasting to be noticed. The points that strike one in this history are—first, these pains, which were severe and then ceased suddenly, with paralysis supervening; and second, the loss of sensation improving. The loss of sensation appears

at first to have been quite complete, and to have affected the whole arm, and there seems to be some question about it having affected the neck. Since, however, she has been treated with the battery sensation has recovered above a line drawn round the forearm two or three inches above the wrist, according to her account. All this, of course, suggests a functional condition; the tongue is not tremulous, there is no nystagmus, the pupils act briskly, and there is some tremor of the eyelids. On roughly testing there is no contraction of the field of vision. On examining the arms she appears to have wrist-drop in the right hand, and a good deal of extensor rigidity of the fingers, well marked in the little finger. The patient can raise her arm to the level of the shoulder, and the movement of the elbow-joint is perfectly free. As regards nutrition there is not much difference between the two arms; there is a great want of tone in the small hand muscles on both sides. On testing for sensation, it is found that there is complete loss of sensation just two inches below the right elbow. Probably if we tested very carefully we should find a line of demarcation right round, and we should mark out what is known as the glove form of anaesthesia, very common with these functional cases. Often you find that the more you repeat the testing the greater are the differences you notice. From the point of view of diagnosis it is better to take the first impression on testing. There is no wasting specially to be noticed, and it must be remembered that the general state of nutrition in these anaemic-looking girls is very poor. It is an extremely good imitation of organic paralysis; there is the sort of smooth condition of skin on the hand, the tapering fingers, and the over extension of rigidity; there is analgesia and anaesthesia up to the line about 2 inches below the elbow; she has no difficulty, however, in walking, the knee-jerks are normal, and she can stand steady with her eyes shut. The patient does not seem to start on being tested for knee-jerks, but says there is a feeling only of pain. I should say it is a typical functional paralysis, and the prognosis one would give is of course a good one. I remember one case which we called functional, however, that nothing seemed to improve. It is important to note in this case that the distribution of the anaesthesia enables us to exclude any lesion of nerve-trunks. From the severe pain at the

onset I thought at first from the description that it might be a brachial neuritis; but if it were you would have the distribution of the anaesthesia following the distribution of the nerves.

Hæmorrhagic Hemiplegia.

The next case is a German æt. 54, a furniture dealer, who last June, while at work, fell back quite unconscious, suddenly, with clonic spasms of the right arm and leg. After the accident he walked to the tram, but on getting home had to be carried to bed. Ten minutes after the attack he had paralysis of the right leg, his speech was affected, and sensation was lost in the right leg,—that means both ends of the capsule affected, supposing it to be of capsular origin. Speech is in the anterior set of fibres, sensation is conducted in the posterior set of fibres, and you notice both were affected. He could walk eight weeks after the attack. The man is extremely anaemic, with a strange waxy complexion. There is complete weakness of the right side of the mouth, independent action of the right eyelid has gone, and the tongue looks on being put out almost as if it were a case of commencing bulbar paralysis. Before the attack he could speak English and German; now he can only speak German, and that very badly. If you get the right amount of tension in the arm you will notice a clonus in the hand; you will notice, too, that he understands English, though he has forgotten how to speak it. There is some rigidity which comes out on quick passive movements of his right arm, and there is some ankylosis of the right shoulder which causes him pain. In walking there is much stiffness noticed in his right leg, and there is also well-marked persistent clonus; sometimes it comes out on the opposite side also, but in this particular case it does not. The knee-jerks are lively. There is a systolic murmur over the pulmonary base, but there is no history of rheumatism. This attack happened a year ago, and, as far as the paralysis is concerned, the prognosis is bad. Any improvement would have taken place before now if he were going to improve. An emotional condition is easily provoked in him, a condition, I always fancy, more marked in right than in left hemiplegia, but which occurs in both forms. This emotional state usually takes the form of uncontrollable fits of crying or laughter. This case shows the difficulty of diagnos-

ing the nature of the original lesion. The initial clonic spasm followed by temporary loss of consciousness, and then after an interval by complete paralysis, would suggest pressure and therefore haemorrhage.

Facial Paralysis.

This is a case of a man who forty years ago had a slight attack of facial paralysis; he is now 60 years of age. The first attack occurred after bathing, and lasted fourteen days, with complete recovery. The history of this second attack is that a week before the onset he had twitching in the lower lip, and then facial paralysis came on during his sleep. There is also some loss of taste from implication of the chorda tympani. In this case the patient had not a high degree of paralysis when he came, and he could half close the left eye, which you now see is improving. In these cases of pressure on the chorda tympani in which you get loss of taste on the same side as the lesion, you often find also a considerable difficulty in getting accurate discrimination of taste on the opposite side, and patients will tell you often that their taste is completely gone for a time. Under iodide of potassium and nux vomica, a blister at the back of the ear, and galvanism, this man has improved much; he has now only a slight weakness in the orbicularis palpebrarum, and is practically well. This slight weakness will probably totally disappear. As regards prognosis, on first seeing the case it is well to be guarded. A small number of cases (? about 5 per cent.) do not recover, but the affected muscles undergo a disfiguring contracture. The more severe cases may take about six months to recover, that is about the time that it takes a cut nerve to regenerate. The milder cases, as a rule, however, take a much shorter time than this.

Ataxic Paraplegia.

This man, æt. 32, comes complaining of numbness and pain in the right leg. Two years ago the pain shot down the leg to the calf. On raising the leg with the knee stiff there is no pain at the hip, practically excluding any sciatic affection; the pupils are unequal but react to light, the knee-jerks are exaggerated on both sides, and there is ankle clonus. His gait is distinctly ataxic; on putting his feet together and shutting his eyes you

notice that he is unsteady, and he cannot walk putting one foot exactly in front of the other in what may be called the toe-and-heel movement. There is also a history of shooting pains in the right leg, and on being closely questioned he admits that he not only cannot hold his water, but cannot tell when he is passing it, indicating of course some urethral anaesthesia. There can also be noticed perhaps a little weakness of the left external rectus muscle. I suppose it to be a case of combined lateral and posterior sclerosis. There is a clear history of syphilis in this case. Ataxic paraplegia is not necessarily a mixture of tabes and lateral sclerosis : in classical ataxic paraplegia the pupils react to light as in this case, and one generally finds that the patient does not complain of lightning pains ; this man does, and of course the knee-jerks are increased with much rigidity, so much so in this case that I thought at the time of examining his knee-jerks that the rigidity was due to the strong voluntary effort of the patient himself. These patients sometimes improve under iodide of potassium and mercury, though it is not easy to see why, bearing in mind the probability that the lesion is a degenerative not an active one.

A LECTURE ON IMPAIRED MOVEMENTS OF THE VOCAL CORDS.

Delivered at the London Throat Hospital, 204, Great Portland Street, March 2, 1896,
By GEORGE STOKER, M.R.C.P.I.

OUR subject this evening is impaired movements of the vocal cords. I shall first allude to the causes which give rise to these conditions ; secondly, the characteristic appearances which are seen in the various forms of impairment ; thirdly, I shall say something about the examination of the larynx and the diagnosis of these conditions ; and fourthly, a few words about treatment.

In order to understand the characteristic appearances which arise in cases of impaired movement

of the vocal cords, it is absolutely necessary that you should have a clear idea of the nature of the structures of the larynx. First of all, the relation of three or four of the cartilages to one another ; and secondly, the exact action which the various muscles or groups of muscles produce on these cartilages.

Firstly, then, there is the thyroid cartilage forming, by the junction with its alæ in front, "Adam's apple;" below that is the cricoid cartilage, consisting of two portions—the ring and the signet, the signet being situated between the posterior extremities of the alæ of the thyroid cartilage, and perched on top of it there are the two triangular arytenoid cartilages. The bases of the arytenoids form right-angled triangles, the longest side of the triangle facing forwards and outwards, and at that posterior and interior angle is the pivot on which these cartilages rotate. These cartilages have not only a rotatory motion, but there is a certain approximating power exercised on them by some of the muscles which govern their actions. The anterior angle is called the processus vocalis, and it is from it that the vocal cord stretches forward, and is attached at the anterior commissure to the junction of the alæ of the thyroid cartilage. It is important to remember these three points : the cricoid cartilages are triangular, they move on a pivot, and they are rotated and approximated by certain groups of muscles.

We will now turn for a moment to the consideration of groups of muscles. In relation to the internal muscles of the larynx, I would draw your attention to what I consider to be the most common order in which they are affected under the various conditions that arise, and I would say here that with regard to anything I may remark to-night I am simply expressing my own views, which are the result of a good many years' observation. In doing so, of course, I may fall into contention with the theories or opinions of other laryngologists.

With regard to the external muscle, which is called the crico-thyroid, and is the external tensor of the vocal cords, it is important to remember how this muscle acts, because it is sometimes a little difficult to understand. When this muscle is called into action, you must remember that the thyroid cartilage is more or less fixed by the muscles which run from the upper part of it to the

hyoid bone, and from thence to the lower jaw. When the muscle contracts it pulls the ring of the cartilage upwards and tilts the signet backwards and downwards, and the distance between the two attachments of the vocal cord is therefore increased, and so the cord is tensed. This muscle, I may mention here, is the one external muscle which is supplied by the external laryngeal nerve. When that muscle is paralysed, whatever the cause may be, the characteristic condition, as seen on examination of the vocal cords, is such as I have indicated by a wavy line in the diagram.

Now we come to consider some of the internal muscles of the larynx. The first that I would direct your attention to is the arytenoideus muscle. It consists of three distinct portions : (1) A transverse, which goes from one arytenoid to the other, covering them completely except at the tips. (2) Two oblique portions that cross each other, running respectively from the base of one arytenoid to the apex of the other. These parts acting together approximate the arytenoids to one another, and close the posterior portion of the glottis.

The thyro-arytænoid lies external to the vocal cord, viz. intimately attached to it. By some the cord is regarded as being the tendon of the muscle. The muscle is attached posteriorly to the processus vocalis, and anterior and outer face of the arytenoid, and stretches forwards to the angle formed by the junction of the alæ of the thyroid. It is sometimes described as a laxor of the vocal cords, but I do not consider that is a correct description of its action. Its action is to straighten the edges of the vocal cords and regulate the chink of the glottis by varying amount of longitudinal tension. This it does by the peculiar arrangement of its fibres, which are extremely intricate, and would require a long time to describe. When this muscle is affected, and it is the second most common, we get an oval opening between the vocal cords,—that is to say, the cords join posteriorly. The only parts that fail to come into the right position are the edges of the vocal cord between the processus vocalis and the anterior commissure, and hence you see an oval opening. In cases, then, where this muscle alone is affected the opening is oval. Sometimes we get a complicated case, and they are not uncommon. When the arytenoideus and thyro-arytænoides are implicated, then we find the combined appearances presented by each

muscle, i.e. a round or triangular opening behind, and an oval opening in front.

We come now to the crico-arytenoid posticus and the crico-arytænoideus lateralis. Now the course of the crico-arytænoideus posticus is that it springs from the posterior surface of the signet of the cricoid, and passes across to the external angle of the arytenoid (processus muscularis) of the other side. You will remember that it is moving on a pivot, and when that muscle contracts, what it does is to pull the point backwards ; and if it pulls the outer angle backwards it must pull the anterior angle outwards, and so it diverges, or is an abductor of the vocal cords. The crico-arytænoideus lateralis has precisely a contrary action. Its origin is in front, from the sides of the ring of the cricoid cartilage, and its action must be to pull the external angle of the arytenoid cartilage forward, and that must push inward the processus vocalis ; so it adducts or brings together the vocal cords. When this crico-arytænoideus lateralis, or lateral adductor is affected, the glottis is open, with a lozenge-shaped opening behind the vocal cords, which is characteristic of paralysis of the lateral cricoid arytenoideus.

Symptoms.—The symptoms which any of these conditions may give rise to, and which should induce us to examine the larynx, are very few, but they are very prominent. I would place the order of the symptoms as follows :—Hoarseness, difficulty of breathing, cough, expectoration. The only special remarks I have to make is with regard to expectoration. It will be necessary for you, when the other symptoms exist, to know accurately the character of the expectoration, because this very often, as I will show you, gives an indication of the cause. It may of itself bring about one form of impaired movements of the vocal cords. In examining our patient we must not neglect to examine the fauces, the soft palate, the tongue, both in quiescence and when it is extruded, because all these, as you may readily understand, will give you an inkling as to what the cause of the hoarseness may be, and you must never omit to test the tendon reflexes. I would advise you very strongly and very earnestly not to commit yourself in any obscure case of this kind to any very definite opinion without a few examinations of the larynx ; because I can assure you that even the most experienced hands get into difficulties by doing so.

Now when you introduce the laryngeal mirror and inspect the glottis, you must always remember that you should do so under two conditions. First of all, when the patient is simply breathing in and out naturally—inspiration and expiration; and secondly, when the patient is asked to phonate or to produce the voice; because there are certain indications which may exist with regard to the paresis or the impaired movement which may be characterised only in the inspiration and expiration, and which you may fail to see or to produce if you tell the patient to phonate at once, asking him to say "a" or "e" as the case may be. In cases of great irritability of the fauces and soft palate, it is absolutely necessary sometimes to use cocaine for the purpose of producing anaesthesia to facilitate examination. We generally paint a ten per cent. solution of cocaine over the uvula and the posterior wall of the pharynx.

We will now consider for a few minutes the various conditions which cause impaired movements of the vocal cords; and what I may say now is simply for the purposes of clinical examination, and not in any way put forward as a scientific classification. My practice is to ask gentlemen to consider these causes under four heads, viz. neuropathic, myopathic, obstructive, and functional. I think those four heads will nearly embrace every form of impaired movement of the vocal cords.

(1) It is certain that cases occur in which the very first symptoms of what afterwards turn out to be paraplegia or tabes dorsalis were neuroses in the larynx. The patients more or less suddenly suffer from great dyspnoea, and almost complete paralysis of abduction is discovered. The cords lying together in the centre, and remaining so necessitate tracheotomy; but such cases are extremely rare. The all-important cases are the peripheral ones; that is to say, cases in which pressure from various causes on either the recurrent laryngeal or the external laryngeal nerves may produce any of the conditions I have alluded to.

I think it is advisable to take these conditions in their usual order of occurrence. My experience is, and I think it is everybody's, that the most common cause is aneurism; of course, more on the left side than on the right. Aneurism affects the recurrent laryngeal nerve by pressure, and produces paralysis. Secondly, I would put down tumours of the mediastinum, and with these I

would be inclined to include syphilitic enlargement of the glands; next, abscesses in or about the neck; and lastly I would mention wounds, traumatic conditions. Cases of aneurism are common enough as far as paralysis is concerned, and tumours about the thyroid very often cause it. Malignant disease of the thyroid is nearly always accompanied by some paralysis of the vocal cord at either an earlier or later stage. With regard to abscesses, one has seen cases in which there is deep cellulitis in the neck, and by pressure during the acute condition, or by the cicatrical contraction afterwards causing pressure on the recurrent laryngeal nerve. With regard to wounds, I have seen one or two cases in which one has had difficulty in finding out the cause of the paralysis, and afterwards it has been discovered that the patient has had a blow on the neck.

(2) Myopathic, that is to say, conditions which have to do with muscles. These conditions, speaking generally, may arise either from some hypertrophy of the muscle itself, something affecting the fibre of the muscle, or something which merely affects the mucous membrane covering it, and which produces an impaired action. The order in which the muscles are most commonly affected is in my experience—

1. The arytaenoideus.
2. The thyro-arytaenoideus.
3. Crico-thyroid.
4. Crico-arytaenoideus posticus.
5. Crico-arytaenoideus lateralis.

These are the muscles which are mostly affected. An ordinary catarrhal condition does not usually affect them. Then there may be syphilis and phthisis. As a cause, specific disease, as I shall point out, forms, according to my experience, a very important factor in the cause of impaired movement of the vocal cords. The infiltration in itself no doubt does often cause some impaired movement of the vocal cords. Then there is phthisis, which also has the same effect. Phthisis nearly always attacks the arytenoid cartilages themselves. In such cases we are accustomed to see an oval or rounded swelling of the arytenoid cartilages, generally of a whitish-grey colour; another characteristic is anaemia of the larynx; in specific disease, instead of the appearances giving the impression of a soft swelling in the larynx, we get the idea or suggestion of a hardened condition

of the tissues. It more often attacks other parts of the larynx than the coverings of the arytaenoid cartilage, and it is generally of a congestive nature. Congestion, in my view as a diagnostic condition, is seen not only in the larynx, but also in the palate and the mouth; in cases of phthisis the pallor of the soft palate is very suggestive, whereas in syphilis the condition of congestion is extremely pathognomonic.

The third head I call "obstructive," and I include in it thickening of the mucous membrane, as differentiated from thickening of the muscles. Then there are growths of various kinds, either malignant or benign; inspissated mucus and foreign bodies. Thickening of the mucous membrane, especially in the posterior commissure of the interarytænoid fold, is characteristic in chronic laryngitis caused by syphilitic disease. I believe that in chronic syphilitic laryngitis you always get thickening of the interarytænoid fold, though you may of course have interarytænoid thickening without syphilis. Then there is inspissated mucus. I think it is correct to put that down, because there are people who have a kind of laryngorrhœa, with a constant discharge of mucus; and a very favourite place for it to collect is in the interarytænoid fold, which prevents approximation of the vocal cords, and undoubtedly induces hoarseness. Foreign bodies are of such rare occurrence, and their nature is so easily determined, that it is not necessary for me to say much about them.

The last cause which I will allude to is hysterical or functional aphonia. I have always been taught, and always believed and understood, that functional aphonia was a form of aphonia occurring usually in the female sex. About the period of menstruation patients get a sudden loss of voice which may or may not under treatment be suddenly recovered. The loss of voice occurs suddenly under certain conditions, but it is not permanent; it is usually recovered from by treatment. It may relapse again, but for a time you can bring it back. I believe and teach that in this condition there is no real paralysis whatever of the abductors of the vocal cords. When you examine the larynx of any patient suffering from functional aphonia, you see the cords fall back, and a triangular opening is left between them. Some people are accustomed to teach that you get an oval opening, but I say that is not functional aphonia, and I maintain that these cases

are due to some paresis of the thyro-arytænoideus; I believe that a triangular opening is an essential characteristic of functional aphonia.

Treatment.—Of course, under all these circumstances, we must attend to general conditions and to local conditions. We cannot remove an aneurism, but we can try to cure it in the usual ways. We may remove tumours of the neck and enlarged glands if causing pressure on nerves. We can do nothing for malignant disease of the thyroid. In neurotic conditions which are the result of blows or wounds we may try electricity. My own view regarding electricity is this: I generally prefer under these circumstances the faradic or interrupted current. Of course, when there is complete paralysis of abduction, and the patient is suffocating, tracheotomy is necessary.

With regard to the myopathic conditions, first of all the local treatment must be directed to reducing those conditions which have produced thickening of the muscle. If syphilis, iodide of potash and bichloride of mercury. If phthisis, the patient may be sent away for change of air, and the usual constitutional treatment applied. In my own view, if there is phthisis in the larynx it is no use to apply irritating treatment. I must mention the lactic acid treatment, which consists in scraping the thickenings and rubbing in lactic acid. Then there is the treatment advocated by Heryng of Warsaw, of removing the tuberculous parts of the larynx. I have tried the lactic acid treatment, but without success, the result I have seen is to create a tuberculous ulcer which one could never succeed in healing. If the myopathic conditions are the result of a simple chronic laryngitis, you must apply the various solutions to the larynx. I think it is better to apply them with a cotton-wool plug—twenty grains to the ounce of chloride of zinc. If you have reduced this condition of thickening, the muscles may still remain weakened, and it is then necessary to apply the interrupted current to induce a stronger and more healthy action of the muscles.

Then with regard to the obstructed forms of impaired movements. If you have hypertrophy of the mucous membranes, you must apply general astringents to the surface, such as chloride of zinc either by spray or brush. As to the interarytænoid thickening, I think the best treatment is to apply the galvano-cautery, but I must warn you that this

is a difficult matter. As to the existence of growths in the larynx, they, of course, may be anything from a mere little nodule on the edge of the vocal cord to a large growth pedunculated, which by dropping between the cords cause aphonia or hoarseness. With regard to the removal of these growths, I do not believe in trying to remove the growth at once. I think it is extremely uncertain what will be removed if the operator tries to do it at the first sitting. I know operators do not always succeed under these circumstances in getting the growth, and I do not think it is a right and proper thing to subject a patient to such a risk. I hold it is the bounden duty of every laryngologist to carefully train his patient by accustoming him to the passage of instruments until he has got his larynx in a tolerant condition; otherwise he may nip off his arytenoid cartilage instead of the growth. I have seen cases in which large portions of the mucous membrane were removed and the growth left behind. This training, is I repeat, absolutely necessary, and it is not with any mercenary motives that I insist upon it. It is the duty of the specialist to say to the patient, "I will charge you so much for this operation, and I shall require you to come here as often as I want you. Of course the sooner I can operate with safety the better for us both; but it is my duty to carefully train your throat before I perform the operation. It may mean six weeks or longer before the operation can be undertaken." With regard to the condition which is produced by inspissated mucus, and so on, inhalations of creosote together with sprays of chloride of iron and chloride of zinc are very useful for restoring a healthy condition to the mucous membrane. When the cords are prevented approximating, then I think these mild sprays, or, better still, applications with pieces of cotton wool, are the best treatment. For applications to the throat, generally speaking, I strongly advise you never to use brushes at all. I recommend the use of a copper wire with the cotton wool wrapped round it. With a brush there is every probability that the epiglottis will be scraped. Some people's larynx is a long way down and some only a short distance, but in either case the copper wire can be bent to the required length.

Case 1.—This patient is the subject of syphilis, and he has what we believe to be an abscess in the anterior mediastinum. Some years ago he suddenly

developed extreme dyspnœa. He was admitted into the Oxford Infirmary, and owing to his aphonia and complete paralysis of abduction he had to be tracheotomized. He left the Infirmary after thirteen weeks' treatment and was a good deal better. He came here suffering from intense dyspnœa, almost aphonic and in great distress. He had complete paralysis of abduction of the left vocal cord, slightly of the right. From time to time all these conditions became accentuated, and relief only came after considerable expectoration of pus. When pus was expectorated, evidently the pressure on the left recurrent laryngeal nerve was relieved. After a few weeks the abscess cavity refilled and all the old conditions returned. The left cord does not move. This man has got pressure on the left bronchus lower down. The right cord is moving properly.

Case 2.—Paresis of the left thyroid arytenoideus. This, from the history of the case, I take to be one which would illustrate a myopathic condition. She has ozæna.

Case 3.—Congenital syphilis—laryngitis. This patient got obstructive impairment from syphilis. When the patient inspires there comes out from the left side of his throat a kind of little red projection or shelf. There is thickening of the interarytenoid fold. Obstructive impaired movement of the vocal cords caused by congenital syphilis of the larynx.

Case 4.—This is a myopathic case. Impaired movement of the vocal cords due to paresis of the arytenoid; the result probably of chronic laryngitis. It presents the usual characteristic, viz. a small triangular opening situate posteriorly, and engaging the posterior fourth of the vocal cord.

Case 5.—Chronic laryngitis. His cords are red, they move freely. The arytenoid stands out quite clear. One cannot say the impaired movement is due to the interarytenoid fold, because the opening does not exist except in the anterior three-fourths of the cord. It is due to paresis of the thyro-arytenoideus.

Periodic Headache.—

B. Fluid ext. ergot	3j.
Elix. quinine	3ij.

La Riforma Medica.

A THURSDAY CONSULTATION AT ST. BARTHOLOMEW'S HOSPITAL.

Amputation of the Breast.

Mr. BOWLBY :—The patient now coming in is suffering from carcinoma of the breast, and the question is as to the advisability of doing any operation at all. She has a large fungating tumour occupying the left breast, and I think the axillary glands are involved to an extent that does not admit of their removal, and the question I raise now is whether it is advisable to remove a part of this disease, recognising that it would not be otherwise removed unless it was because of the haemorrhage which is taking place from this fungating mass. Some time ago I brought a patient in here, a woman with a fungating tumour in the breast, bleeding excessively, and I raised the question then as to the advisability of removing the tumour from the breast in order to give her some relief from the haemorrhage. That is the question to-day. I myself shall advise the operation. I cannot remove the whole of the growth, which has infiltrated beyond the clavicle.

Mr. THOMAS SMITH : I should remove what I could, though the patient's end will not be long in coming, yet the relief from this fungating sore and the haemorrhage, will be of great comfort to her.

Mr. LANGTON : I should strongly advise an operation, and I should remove one or both pectoral muscles and sweep out the glands in the axilla.

Mr. BOWLBY : There is a lump, probably a lymphatic infiltration, above the clavicle.

Mr. LANGTON : I should try to even remove that.

Mr. HOWARD MARSH : You will stop the haemorrhage by the operation, but I think it is a too widely diffused growth for the removal of the axillary vessels. She clearly has a very widely infiltrating tumour; and I do not think myself that you will get any margin which will be of healthy tissue, and you would only have early recurrence, and that would make it inadvisable to undertake a very extensive operation.

Mr. WALSHAM : I take Mr. Langton's view and should make a clean sweep of the breast and glands. She is a very thin patient, and provided

there is no visceral trouble it could be done with very little risk.

Mr. HARRISON CRIPPS : I should operate on the breast myself; that certainly ought to be removed. There is a gland above the clavicle, but it may not be connected with the mass in the axilla. Under an anaesthetic when you have made your incision for removal of the breast, it might seem practicable to go on to the removal of the glands.

Mr. BOWLBY : I am glad to hear that the disease is, in the opinion of my colleagues, a little more removable than I thought myself. It is in a condition in which one would not ordinarily advise operation in malignant disease, but the haemorrhage is the cause of the operation being done, even though the disease will be not completely removed.

Carcinoma of the Oesophagus with Secondary Deposit in the Glands of the Neck.

Mr. HOWARD MARSH : This patient, who is 45 years of age, has a hard glandular swelling on the side of his neck as large as a small orange. Four and a half months ago he noticed a small lump which has increased steadily, and forms now a considerable mass. First we must consider the diagnosis, and then we will determine whether it would be advisable to submit him to an operation for the removal of the swelling. The patient has lost flesh very rapidly, is now thin and weak, and looks, as you will observe, very ill. He has paralysis of the left vocal cord, there is difficulty in swallowing, and he brings up a good deal of mucus from his oesophagus. I have not examined him myself yet with an oesophageal bougie, but Mr. Marshall found on attempting to pass one that it gave the patient so much distress that he did not persist in the examination, so we have no clear indication of the state of the oesophagus from that source; but from what I learn about his history, and especially with this paralysis of the vocal cord, it appears probable that the case must be one of secondary carcinoma, the primary disease being in the oesophagus. The first thing he was conscious of was a swelling in the side of his neck. I think there can be no doubt that this must be malignant; but whether it is secondary to disease in the oesophagus, or a lymphosarcoma, occurring primarily in the neck, I do not feel quite sure. Mr. Marshall in passing the

œsophageal bougie seemed to come to a definite obstruction about twelve inches from the teeth, but that is not quite certain. I now leave the case for the consideration of my colleagues, expressing myself the view that it is probably a secondary mass of carcinoma, the primary disease involving the œsophagus; and under these circumstances I do not consider any operative treatment should be adopted.

Mr. THOMAS SMITH: I think this case is undoubtedly malignant, and I am rather disposed to think that it is primary disease of the glands, the growth seems so separate from the pharynx and the larynx. There is the same shape as glands would have with primary carcinoma occurring in them. I think you could do nothing by an operation.

Mr. LANGTON: I share with others the opinion that this growth is malignant in its nature. There seem to be two distinct sets of glands involved, one moveable, and the other, the larger set, more deeply fixed on the outer side of the neck. Whether due to primary disease in the œsophagus I do not know, but I should oppose the idea of it beginning in the œsophagus. I should, however, pass an œsophageal tube under an anæsthetic in order to ascertain if there is any stricture. But under no condition would I advise any attempt at removal in this case. The man is infected with the disease too extensively to admit of an operation.

Mr. HARRISON CRIPPS: I agree with the view that this disease is malignant, but do not consider there is sufficient evidence to regard it as secondary to disease in the œsophagus; there is difficulty in swallowing, but this appears to have occurred quite recently. I do not think it possible to successfully remove the disease.

Mr. HOWARD MARSH: I may just mention that a few months ago I had under my care a case of spontaneous fracture of the femur, which, as post-mortem examination showed, was due to a deposit of carcinoma secondary to a primary disease of the œsophagus, and in that case there was no difficulty in swallowing.

Mr. BOWLBY: I think it is a cancerous growth of the glands secondary to a primary growth in the œsophagus. A lympho sarcoma is not likely to cause paralysis of the vocal cords. I had a man last year under my care with a doubtful tumour in

the neck; there was no evidence of its implicating the œsophagus, and he swallowed quite well up to the time of his death. At the post-mortem, however, there was found a growth in the œsophagus, but it was not obstructing it. Mr. Butlin brought in a man some time ago showing paralysis of the sympathetic nerve, and that turned out to be a secondary disease to a growth in the œsophagus, and in that case also there was no difficulty in swallowing.

Mr. HOWARD MARSH: This patient now has some difficulty in swallowing.

Mr. LOCKWOOD: I think it is a case of extensive infiltration, and the disease tracks down into the anterior mediastinum; there is also some venous obstruction to be noted. You cannot do anything, in my opinion, in the way of operation.

Tubercular Disease of the Epididymis.

Mr. THOMAS SMITH: This patient is 35 years of age, and his occupation is that of a sea butcher. Three years ago he noticed a small pimple, which afterwards became a swelling after injury to his scrotum while riding. He noticed a slight pricking for some time; after this a lump came which did not increase in size for twelve months. The lump discharged a watery fluid. There is on examination some deposit in the epididymis, some scars and fistulous openings in the scrotum, where discharge occurs. The testicle appears free from disease, the vas feels like the stem of a tobacco pipe, giving one the idea that it is affected with a tubercular deposit. The man is taking iodide of potassium and steel wine, and he is very anxious to avoid any operation. I have brought him in to see if any of my colleagues can suggest anything short of operation.

Mr. LANGTON: Is there any frequency of micturition?

Mr. THOMAS SMITH: No. The epididymis seems full of tuberculous deposit, which occasionally softens and discharges itself through the sinuses in the scrotum.

Mr. LANGTON: Is there any disease in the chest? Has the man suffered from cough?

Mr. THOMAS SMITH: No. The man has a nodular swelling in his epididymis, which now and then apparently breaks and discharges through the various sinuses, and having his vas deferens thickened and hard and with a deposit in it, it is reasonable to suppose it is tubercular; and the reason

why it has not become larger is, I suppose, from the fact that it now and then softens and discharges. Should one do anything by way of operation? I think it would be better to have the testicle removed, but he is very adverse to that. There is, of course, an alternative in the way of operation, and that is to lay it open and scrape it, and rub on emulsion of iodoform. Whatever might be done by way of an operation I think it would be advisable to clear the vas as much as possible. The man believes himself to be very well, and it is very hard to persuade him to submit to an operation. He says he can pursue his trade; and the only thing he complains of is that he has a little pain, if he walks about much, in the perineum.

Mr. LANGTON: I quite agree with you that it is tubercular disease. The testicle is perhaps a little softer than it ought to be. It is functionally incompetent, and the whole of the epididymis seems to be involved. The best thing would be to have his testis, epididymis, and vas removed, as high up as one can. One need not assume that there is disease of the vas itself, the disease is probably not so high up as it appears to be. Perhaps it might be scraped, as you suggest; but the better plan would be to have the testis and epididymis removed.

Mr. HARRISON CRIPPS: I think it must be a case of tubercular disease involving the testis, and the epididymis, but I should not advise excision of the testicle. He has undoubtedly an abscess. This I should open and thoroughly scrape the walls, and believe it might heal up without the necessity of castration. So far as the cord is concerned I do not think that there is any evidence that that is tuberculously infiltrated. I have felt some cords very much thickened; and yet when this primary disease in the testicle has been scraped away, this thickening has slowly cleared up.

Mr. LOCKWOOD: It appears to be tubercular disease in the epididymis, and accordingly I should suspect the thickened part running up to the ring on the vas to be of the same nature. I do not think it is a case for local treatment, I should myself advise complete removal.

Mr. THOMAS SMITH: It is not very easy to persuade the man to allow a surgeon to take away his testicle, for he does not believe himself that his testicle is of no use to him.

Nævus, suitable for Operation.

Mr. BOWLBY: This patient is sixteen years of age, and you see that she has a very extensive nævus occupying the whole of the right side of the cheek, the superior maxillary region, and extending through the whole thickness of the cheek, and it is seen as a considerable swelling in the inside of the mouth. I will direct your attention to the extent to which this quite soft growth has thrust inwards the whole of the teeth. This bulging mass by its pressure has compressed the jaw-bones on this side, flattening and pressing them in. There is a scar running across the nævus, which is bluer and evidently more venous than the rest of the diseased part. That is the result of an earlier operation which appears to have been done some time ago, and which seems to have done some good at the time. It was a partial removal perhaps, and treatment with perchloride of iron. It appears to have been done by Sir George Humphry. Her mother has brought the child because of the increasing deformity. The question is, can anything be done? I may say that the question that I really raise is as to the advisability of doing anything in the way of a cutting operation where the skin can be lifted from the nævus. The advisability of doing something is very evident. What can be done to improve it materially? I myself should advise some operative procedure. What I was thinking of doing was to see if the skin could be lifted up and separated from the subjacent growth. The proceeding involves considerable risk from haemorrhage. I should advise, on the whole, an operation of lifting up a flap of skin, and removing a considerable part of the growth. The part next the mucous membrane might be subsequently dealt with by electrolysis if the first operation proved satisfactory. Of course electrolysis might be tried first, but my only fear is that the electrolysis treatment would interfere considerably with the tissues in the case of a subsequent operation, and I am inclined to advise the patient to have the nævus operated on first, and to have some of the growth cut away, and subsequently destroy anything that is left by electrolysis.

Mr. THOMAS SMITH: If I could not get Dr. Lewis Jones to undertake to cure that by electrolysis I should do what Mr. Bowlby says; but I should be very particular to clamp the lower part

of the cheek and take away as much as I could of the growth; but the circulation must be very carefully controlled by clamps, or else the operation will be very dangerous. For myself, I should attempt it if the haemorrhage was carefully controlled.

Mr. LANGTON: I should recommend some operation; the naevus is growing, and the part operated on some years ago is really the worst part of it all, for it involves the skin, and along the scar there is, as you see, a considerable amount of naevous tissue in the skin itself, which is probably fed by at least three sources, the facial vein, the jugular, the angular vein, and probably also one of the branches corresponding to the transverse facial. I do not think it is a fit case for electrolysis myself because it is fed by such large venous trunks. I should ligature the facial vein and other veins, firstly, and then I should dissect it out and get away what I could, and then, perhaps, I should deal with the upper part of it where the skin is free; perhaps the naevus might contract subsequently, and you might then be able to deal with it by electrolysis.

Mr. HARRISON CRIPPS: With the free use of pressure forceps I think you might control the haemorrhage, and that would be the great difficulty. I should begin from below and work upwards, and secure the veins which would be affected by the operation. As to whether it would be best to remove this growth at one operation must depend on what occurs at the time of operating; if there is not dangerous bleeding, it might all be done at once; if there is, a portion might be left to deal with subsequently.

Mr. LOCKWOOD: I should not, on any account, begin by electrolysis. Supposing the electrolysis were to fail, the tissue would be in such a condition that you could not use other methods. I should choose a cutting operation, and turn down a flap and cut away the naevus as much as possible, and I think you could get away a great deal, and then ligature the rest of the growth.

Gall-stones.

Mr. LOCKWOOD: This patient is a woman æt. 60, who has had during the last year four or five attacks of jaundice. I saw her a few weeks ago when she was, however, only suffering from a slight attack. Before the attacks of jaundice she has violent pains in the upper part of the ab-

domen, then she becomes jaundiced, and after suffering from pain for some time it passes away. I feel sure that I can feel in her abdomen an enlargement in her liver, and I think also I can feel her gall-bladder with some stones in it. I propose to open the abdomen and empty the gall-bladder and remove any stones from the cystic or other ducts. She says this disease makes her life a burden to her. I do not think that the present condition is dangerous, but from my conversation with her I am sure the disease is a grave inconvenience to her, and she is prepared to undergo the risk. I do not myself think that the risk is a very great one, and with my colleagues' consent I propose to undertake it.

Mr. THOMAS SMITH: I think myself you are quite justified in doing so: I do not myself feel the gall-bladder, but I am sure that what one feels is rather more resonant than gall-bladders usually are. You will be of course justified in making the examination by an operation.

Mr. HOWARD MARSH: I do not myself feel the gall-bladder very clearly, but I feel a distinct, resisting swelling, which is probably the gall-bladder. I think it is quite justifiable to make an examination.

Mr. HARRISON CRIPPS: Have you seen her yourself in any severe attack?

Mr. LOCKWOOD: I have only seen her in a very slight attack.

Mr. HARRISON CRIPPS: Affection of the cystic duct would not itself produce jaundice, and if in the common duct you would find the gall-bladder enlarged. I am inclined to think the hepatic duct is obstructed, and I cannot satisfy myself that there are any gall-stones to be felt. If the patient consents you are perfectly justified in making an incision, and you could examine the duct after the gall-bladder has been investigated.

Mr. THOMAS SMITH: We all mean of course that the disease is in the ducts, and that they should be examined.

Mr. BOWLY: I think the contraction of the gall-bladder on the stones causes the pain I know cases where pain has been caused in that way. I think it is not cancerous disease, because there is not much jaundice. In these cases of cancer the patients are very much and very deeply jaundiced. I should myself expect to find stones in the bladder.

THE CLINICAL JOURNAL.

WEDNESDAY, JUNE 17, 1896.

A CLINICAL LECTURE ON SOME SURGICAL SEQUELÆ OF CHRONIC NERVE DISEASE.

Delivered at the Central London Sick Asylum, in connection with the London Post-Graduate Course,

May 7th, 1896.

By JOHN HOPKINS, F.R.C.S.

Surgeon to the Hospital.

LADIES AND GENTLEMEN.—The first case or two which I propose to show you to-day will be illustrative of perforating ulcer. They cannot strictly be called perforating ulcers at the present time, as there are no open sores ; but the term applies now to a very wide range of clinical and pathological facts, so that one may feel justified in using it even when there is no perforation or ulcer. I may say that the term originated when the pathology was not understood.

In the year 1852 Nélaton described this condition, and in the following year, or about the same time, Vésignié advanced the theory that it was a form of plantar psoriasis, and suggested the name *mal plantaire perforant*. In the year 1855, or three years later, Leplat dealt with the same subject, and attributed the condition to pressure ; and no doubt in this he was correct, but it was obvious that some other cause operated. In 1863 several cases were published in which there had been found a degeneration of the blood-vessels ; accordingly the explanation then advanced was that the disease was due to imperfect nutrition due to lack of blood. In the following year—1864—it was pointed out how much resemblance there was between perforating ulcer and the condition found in anaesthetic leprosy, and in 1871 Estlander advanced the theory that it was really anaesthetic leprosy ; and seeing that this latter disease had at one time prevailed in Europe more widely than it

did when he wrote, he came to the conclusion that it was the last remaining vestige of the disease. The remarkable resemblance that it may bear to perforating ulcer is shown in the illustrations of Mr. Clinton Dent, which I will pass round ; they are in the 35th volume of the Pathological Society's "Transactions." Both feet are very much shortened, —the toes, in fact, resting on the tarsus. That was a condition brought about by repeated perforating ulcers, and appears to have been an inherited condition, as it occurred in the maternal grandmother and two of the patient's own brothers. The theory I last mentioned was very quickly abandoned, for in 1872 Poncet, in studying this theory, discovered that there was some degeneration of nerves in perforating ulcer, and from that time pathologists got on to the right track ; and subsequently, all the cases which were recorded were found to be associated with degeneration of the sensory nerves in relation to the part.

The term "perforating ulcer" was originally applied to a condition which may be briefly summarised as an opening in the sole of the foot, which, when probed, was found to lead down into the deeper tissues by a sinus, at the bottom of which, bone in a condition of caries was sometimes discovered. There was *always* a thin foetid discharge, and the feet were bathed in perspiration. Nothing was then known of the pathology ; the term simply described the condition.

Now one would infer from the term "perforating ulcer" that, to begin with, there is an ulcer in the sole of the foot, which, by ulceration, passes deeply into the tissues. But that does not at all describe the actual process. It does not begin as an ulcer, and when it does perforate it does not do so by ulceration in the common acceptation of the term. As a matter of fact, it is nothing but a large corn with a suppurating bursa beneath it.

The numerous cases of this disease which have been published show that it may be associated with a large number of nerve diseases ; it may occur in peripheral neuritis, and that may be primary or secondary. It may be secondary to diabetes. Dr. Stephen Mackenzie tells me he has

seen a case of perforating ulcer in peripheral neuritis which occurred in diabetes. It occurs in a variety of cord affections. A case was published in the Pathological Society's "Transactions" by Mr. Pepper and Dr. Silcock, vol xxxvi, in which there was a general degeneration of the spinal cord. I hand you the plate of this, which also shows the nerves in various stages of degeneration. Another case, published in vol. xxxvii of the same "Transactions" in the following year by Mr. Bland Sutton, illustrates the same condition in a civet-cat; and between reading and publishing that paper the author says he saw several cases of perforating ulcer in animals in the Zoological Gardens, which were all the subjects of paraplegia. Dr. Ogston many years ago published a record of a case of perforating ulcer occurring in an adult, who had recovered spontaneously in early life from spina bifida. The foot was affected with anaesthesia and talipes on one side, and there was a depressed scar in the centre of the back over the spine, in which the spinous processes could not be felt. The perforating ulcer was no doubt due to degeneration of the nerves in consequence of the spina bifida. Perforating ulcer has occurred in slowly advancing paraplegia following an accident, but the commonest condition in which it is found is posterior sclerosis. We know that in posterior sclerosis there is generally some degeneration of the sensory nerves—a condition which, as I have already pointed out, is found to be always associated with perforating ulcer. I have not seen it recorded as occurring in general paralysis, but I have no doubt it does occur in that disease, at all events in some of those cases which are affected with tabes; and there is a similar condition occurring under like circumstances, which I will refer to again later on. I have several times seen in early general paralysis a suppurating corn on the back of a toe, perforating the joint, leading to disease of the joint or loss of the toe. In addition to degeneration of the nerves and the peculiar tendency to corns that we find in these cases, the bones and joints are often affected, as well as the nails and hair. The cartilages of the joints are softened and worn down, and there is a tendency to what may be termed compensatory hypertrophy in the growth of new cartilage and bone in the neighbourhood, so that the bones may be welded together. I may mention also that the subjects of perforating ulcer

are predisposed to erysipelatous inflammation in the affected limbs.

We will now look at the cases.

This man's pupils react in the act of accommodation, but not to light; the patellar reflex is absent, and if he were out of bed you would discern some, though not very marked, ataxia. There is some degree of anaesthesia in the sole of the foot, and he experiences lightning pains. The condition I wish to show you on the sole of his foot is simply a corn, which he has recently whittled away a good deal. You see a large circle of thickened epidermis, with a hard central core which, however, rapidly varies in aspect, owing to the quick growth of epidermis. A week ago the centre was hard and dry, with a deep sulcus, which, if continued round, would have completely separated the central hard core from the ring of thickened epidermis around it for a depth of two or three lines. The corn in this patient has several times been an open one, the hollow being large and ragged, allowing the probe to pass some distance under the thickened border. The way in which this ragged cavity closes is not by a growth of epidermis from the bottom, but from the periphery, gradually narrowing the opening until it becomes like a pin-hole; this progressive growth towards the centre continuing, finally closes the opening, and forms this hard, thick central mass. This, contracting, separates from the ring of softer thickened epidermis, thus forming the above-mentioned fissure. The patient has come here on two or three separate occasions with this open corn, accompanied by diffuse inflammation of the foot. If he went out again the same condition would recur. If the foot perspired very much the corn would naturally become softer, and if he had sufficient sensation in the foot to feel the inflammation going on in the deeper parts of the corn he would cut it, and let out the contents of the bursa. If he had not enough sensation for that, the contents of the bursa would suppurate and discharge, either at the side of the foot, or on the dorsum, or between the toes. Then, if the corn were opened, he would have a genuine perforating ulcer. On his other foot you see two large corns. That is a condition which is not usually found in a patient who has been in bed some months, for in that time the normal epidermis peels, and any corns which may have existed disappear. Corns

due to degeneration of nerves do not seem to diminish, however much they may be rested. The man complains of pain in the centre of that corn that lies under the big toe-joint, and you are able to see that there has been haemorrhage beneath it, which has stained the deeper layers of epidermis. If this corn were cut, these deeper layers would be found softer and tending to crumble.

If you saw this next patient walking, you would find he has no ataxy. His pupil reflex is normal. His patellar reflex is good on both sides. The ordinary clinical tests used show practically no anaesthesia in the skin, but inquiry elicits the fact that he suffers from lightning pains. I had no idea that the man had any such nervous affection when he came here with bronchitis at the beginning of the winter. Two months ago he drew my attention to the fact that the back of his foot was red and swollen, but he did not feel any pain. I found an abscess pointing on the dorsum between two of his toes, and on opening it a probe passed straight through into the middle of the corn I show you. He had been walking about the ward with a suppurating bursa under his corn without being aware of it. I ascertained that he had suffered from lightning pains at times for two or three months. On his other foot there are two areas of thickening. A perforating ulcer can be readily healed, and these two cases have done so. They had each a daily foot-bath of creolin. I did not trouble to open this corn, but rested content with the exit through the dorsum. All that can be done for these two cases has been done. There is no doubt the time will come, if they get about and to work again, that they will have a recurrence of the trouble. Every now and again one meets with cases in which there is an astonishing loss of sensation to pain, without the abnormality having been suspected. The fact is sometimes brought to notice in a very unusual way. For instance, a man breaks his femur, and when an attempt is made to put it up after the accident no pain is experienced. Two and a half years ago I had a case in which a femur was fractured, and the patient was insensitive to pain in the tissues in the proximity of that fracture. A long splint was put on, and the arm had to be moved about a good deal to accomplish this, yet it was not until two or three days afterwards that I discovered he had a fractured clavicle. The

patient was not aware of it, and had been making what use of his arm he could while lying in bed. There was nothing beyond what I have related to indicate that he was the subject of chronic nerve disease.

Another case of analgesia which I had was a man suffering from fistula. On making an ordinary examination to discover its extent we found he had no sense of pain, therefore I proposed to cut the fistula without administering an anaesthetic. He consented, and told me he did not feel it. The operation was not sufficient to cause the fistula to heal properly, therefore I scraped it with a spoon, again without an anaesthetic. He said he felt that, but it gave him no great pain. Finding he could bear operations without suffering pain, he suggested I should cure him of a Dupuytren's contraction of one finger. I inserted the knife, by Mr. Adams' method, between the skin and fascia, and scraped through the fascia with the point of the knife until it gave way; but he did not wince or change colour, and I do not think he felt it much. The remarkable fact about this is that the ordinary clinical tests afforded no evidence of analgesia or anaesthesia. Taking that in conjunction with the fact that perforating ulcer may be present without any other signs of nerve disease, the obvious conclusion is that profound and advanced nerve disease may exist without any clue to it being afforded by the usual clinical tests; therefore perforating ulcer—using the term in its broadest signification—should be sought for in all cases where nerve disease is suspected.

The next patient I have to show you, a man, is the subject of a very chronic form of ataxia. In these cases where there is perforating ulcer, the motor symptoms, as a rule, are not very marked. He has a little suggestion of ataxia in his gait at times, and that is all. He has been here continuously for eleven years. I will detail the points in his case, though some of them are not directly related to our subject. He has had syphilis, and since his stay here he developed, somewhat rapidly, an erythematous patch across the bridge of the nose and cheeks. This very rapidly ulcerated in small spots, and it spread until it covered the nose and extended some distance further upon the cheeks, so that I thought there was going to be an extensive ulcerating area. The ordinary treatment by mercury and iodide of

potassium did not seem to touch it, but on giving him a mixture containing iodide of iron, iodide of potassium, and tincture of iodine, the condition quickly improved and the ulcers cicatrised. On the front of his left leg you can see a large white scar, surrounded by pigment, probably due to the specific trouble, though you may see the same pigmentation around an old chronic ulcer where there has been no syphilis. The skin of that scar is fairly supple and not very adherent to the parts beneath.

I remember one old woman who had a huge white scar extending over two-thirds of the front of the leg; that also was a good sound scar, only slightly adherent, and she said she had been free from ulceration for twenty years, though the condition had lasted for twenty years before the ulcer healed. Such cases are encouraging, and I believe that proper attention to a scar after the ulcer has healed will go a long way towards permanently curing a large percentage of chronic ulcers. The next thing we notice about the patient before us is that the right limb is much larger than the left. He has had diffuse inflammation a great many times during the eleven years he has been here. The cause of this repeated inflammation and consequent solid oedema is a discharging sinus in connection with chronic disease of the ankle-joint. He has lost the great toe also, which he says was due to erysipelas, and no doubt he had that disease at the time, but probably the actual opening of the joint was due to a suppurating corn. Or it may be that his joint had already degenerated from the ataxy he suffered from, and therefore was rendered vulnerable to the erysipelatous micro-organisms. After getting about again he once more had diffuse inflammation of the foot, and that led to suppuration of the ankle-joint. He came in with that joint greatly distended, the ligaments apparently soft and stretched, and allowing the parts around to bag outwards in consequence. There was little or no evidence of pain in the deeper tissues; nor much on evacuating the inflammatory products by an incision. Some years ago I removed a man's toe on account of a diseased joint, and he subsequently had erysipelas of the foot, caries of the metatarsal bone, and suppuration of the left wrist. The left wrist was incised, and fortunately got well; the metatarsal bone was removed under very strict antiseptic precautions. After removing

the bone and scraping the tissues, these were thoroughly scrubbed with chloride of zinc, 20 grains to the ounce; then with 1 in 20 carbolic, followed by 1 in 1,000 perchloride lotion, so that we thought we had guarded against the possibility of a recurrence of diffuse inflammation. But notwithstanding this he had a recurrence of the inflammation after the operation, with the result that he had suppuration in the other wrist, which led to the loss of the hand. Fortunately there was only a little suppuration in the wound following the amputation, and no other joint was affected. Now, that man had no definite signs of chronic nerve disease except tremor; no anaesthesia, no affection of the pupils or reflexes, nor any of those signs by which one judges of the presence of chronic disease. As I have said, he had tremor; his nervous system was not well balanced, and one other condition pointing that way was that his hands and feet perspired profusely at times without obvious cause. There is no doubt in my mind, however, that he had some ill-defined chronic nerve affection, and that his tissues were in consequence of that particularly susceptible to the inroads of micro-organisms. I believe the affection of the two wrists was no mere accident, and the explanation that suggests itself is that the two joints were singled out because they were equally degenerated. With regard to the loss of this man's toe, that is not an uncommon event in these cases; I have also seen several cases of lost toes in early general paralysis, in which the condition appears to be exactly the same as in perforating ulcer. The pressure on the skin causes a development of epidermis in excess, and this produces a sort of compensatory hypertrophy of the papillæ immediately around, while causing atrophy of the skin beneath. A bursa is developed; sooner or later suppuration takes place in it, and as the pus cannot escape through the thickened epidermis it passes into the joint. The reason the pus enters the joint is doubtless because it is not detected in time. One speck of pus will cause most excruciating pain in a normal person, and therefore lead to its liberation, but these analgesic patients, hardly conscious of anything wrong, continue to subject the part to pressure by wearing boots and walking about. Thus the pus is permitted to burrow deeper into the tissues instead of passing to the surface, with the results I have mentioned.

The joints are no doubt profoundly altered as a result of nerve disease, and we know they are very prone to invasion by pathogenic organisms.

In tabes, as is well known, you may find a Charcot's joint, say, in one knee, without evidence of Charcot's disease in any other joint in the lower limbs, though on post-mortem examination one may find that in most of the other joints there is very considerable change, such as ulcers or depressions in the cartilages. Or the cartilage may be fibrillated, softened, and worn down to the bone by the movements of the joint. Again, you may find cartilaginous bosses in the neighbourhood of the joints, and osteophytic growths in ligaments. Not only the joints but the bones and ligaments are softened, and are readily fractured, and I suspect that was the case in the man I spoke of who fractured his clavicle and femur. The ligaments give way in these patients with the exercise of very little force. This was well illustrated in a case I had many years ago, which was related in the Pathological Society's "Transactions." He was the subject of ataxia, and, while turning in bed, fractured his femur obliquely between the middle and lower third. One would have thought that the force exerted whilst turning in bed would be all expended in producing this fracture; but such was not the case, for he sprained his knee-joint as well, and there was an abundant effusion into it.

In regard to the invasion of degenerated joints by micro-organisms, such a result as abscess of the knee-joint from what would prove a trifling injury in normal health, may occur in paraplegia. If by chance one has the misfortune, by the employment of a hot-water bottle, to excite a blister on a paraplegic limb, one will perhaps find, without much warning, that suppuration is spreading deeply throughout the limb, or perhaps the first evidence of mischief, beyond a febrile condition, is a collection of pus in the joint. In ordinary cases of erysipelas the joints are not invaded, or very seldom. One other point about the case is that he has a large corn on his left foot, though he has done nothing but tramp about the wards, and very little of that; the nails are also affected in the usual way, being ridged, cracked, and breaking off in fragments.

To summarise the conditions above indicated, we have first, degeneration of nerves, leading to or associated with degenerative changes in the

skin and its appendages, in the bones, ligaments, and joints, and probably in all the tissues throughout the parts supplied by these nerves.

Second, failure of the parts concerned to resist ordinary pressure, friction, and strain, with the result that the cartilages are worn, and the skin is excited to an excessive formation of epidermis at points of pressure, followed by atrophy of the underlying skin. In the case of both cartilage and skin there is at the same time excited a lame attempt at compensatory hypertrophy around the degenerated areas.

Third, failure of the tissues to resist the inroads of micro-organisms, with the result of suppurating bursæ, perforating ulcers, caries of bone, recurrent attacks of erysipelatous inflammation, together with suppuration in and destruction of joints, leading to loss of toes, or it may be more or less of a limb.

There are two other cases I may fitly show you in connection with the foregoing. The first one I show you is a woman well on in life. You will notice, looking at the back of the left hand, that her fingers are everted, and the heads of the metacarpal bones are enlarged considerably. There is evidently some arthritis here, and there is the same condition in the left shoulder. There is a depression at the insertion of the deltoid; the axis of the humerus is not in its normal position, and on moving the arm you can hear crepitus. There is no doubt a wide glenoid cavity, due to partial absorption of old, together with formation of new bone, and the head of the bone rests near to or against the coracoid process. In this case there is evidence too of former whitlow, and you will perceive a scar here and there—all evidences of analgesia. A scar on her hand was produced by holding a hot inhaler, which burnt her without her knowing it. The analgesia is unilateral, and I will demonstrate that by ice and a hot spoon. You see there is no loss of sense of touch, but a loss of power to appreciate pain and heat. That is a condition found in only one disease, namely, syringomyelia, in which the sense of pain and of heat and cold is separated from that of touch. Syringomyelia is due to the formation of a cavity in or near the central canal of the spinal cord; this cavity enlarges, and there is an accumulation of fluid in it, which presses upon the neighbouring parts, with the result that in addition to the analgesia and arthritis of the left upper limb in

this case we find some affection of the lower limbs, due to pressure. If the pressure be in the direction of the lateral columns we find a spastic condition; if upon the posterior columns the condition is ataxic: in the case before you the right limb is drawn up and the foot inverted spasmodically, and there is very considerably increased tonus in the limb. She can lift one leg from the bed, but not the other. When she came in she could walk, and complained of stiffness in the limbs, difficulty in getting them to move, and that they drew up spontaneously while she was in bed. The morning after arriving she fell down and hurt her shoulder, but it was not observed for a few days,—in fact, I did not hear of it until a week afterwards, and when I made the ordinary routine examination I found the head of the humerus in the axilla. I reduced it, and put her up in bandages; but, however fixed, she always managed to wriggle her arm out at night, because it was much more comfortable without any bandages. Moreover she used her arm as if nothing was the matter with it, and raised herself in bed by means of it. Following on the dislocation, inflammation of the deeper tissues set in, apparently excited by the contents of the joint, for on making an incision where there was some redness and probably inflammatory products, and passing in abscess forceps, I opened a cavity into which the finger could be passed towards the axilla, and from which I removed several melon-seed bodies, which had evidently escaped from the joint. This proves that previous to her accident she showed evidence of arthritis in the joint. She has been here four or five years, and her condition is slowly progressive. After the accident she took to her bed, and I do not think she has been able to walk since.

One other case I would like to show you—a woman the subject of ataxia. She has some ptosis of the right lid, and some strabismus. The pupils do not react to light; there is no knee-jerk, but she is able to raise her feet from the bed. She is also subject to lightning pains. The disease is considerably advanced in her, and you will perceive considerable wasting of the leg muscles. It is primarily a case of ataxia in which the disease is now invading the anterior horn. The interesting part of the case is that she has a Charcot's joint in the spine; but I must ask you

not to touch her, as it causes her prolonged distress. There is an abnormal bend at the junction of the lumbar spine and the sacrum, and abnormally free movement there. When she moves you are able to hear a very loud crepitus there. The whole upper part of the body is thrown forwards, and the margins of the ribs lie below the anterior iliac spines. I believe this is the only case of the kind to be seen. One similar case has been reported in the transactions of a Bordeaux society, for a reference to which I am indebted to Professor Clifford Albutt, and that is the only other I have heard or read of.

A CLINICAL LECTURE ON FRIEDREICH'S DISEASE.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London,

On May 20th, 1896,

By J. A. ORMEROD, M.A., M.D., F.R.C.P.,

Physician to the Hospital, and Assistant Physician to St. Bartholomew's Hospital.

GENTLEMEN.—I fear that the subject I have announced for to-day's lecture may not be regarded as one of very general interest, but my reasons for the choice are, first, that the disease is not so uncommon as to make it unlikely that you will see cases of it by-and-by; and secondly, I happen to have several patients accessible to me who are suffering from this complaint. So that I shall not merely lecture on the disease, but shall also be able to exhibit it as it occurs in nature.

The complaint is generally known by the name Friedreich's disease, from Friedreich of Heidelberg, who first, and very accurately, described it. Occasionally it is called hereditary ataxia. Some object to the first name on the ground that it is inexpedient to name diseases after their discoverer; while the objection to the second is that there happens to be another, and more recently described form of hereditary ataxia.

I will now briefly mention the symptoms which characterise the disease, taking first those which are most common as described by Friedreich.

The leading feature is ataxia, which first affects the walking, then extends upwards to the arms. This ataxia is commonly of the "cerebellar" type, *i.e.* an irregular, staggering gait, and not characterised by a flinging about of the legs as in tabes. It resembles tabes, however, in that it begins with unsteadiness of gait, the unsteadiness proceeding to the arms; and that the knee-jerks are abolished. The second main symptom, which usually does not occur until some years after the onset of the disease, is an affection of speech, a drawling, hesitant, and sometimes explosive utterance. The third feature described by Friedreich is nystagmus; this also, as a rule, occurs quite late in the disease, so that you cannot exclude Friedreich's disease by excluding nystagmus. There are other symptoms besides these chief ones, one of which is that along with the ataxia there is what I should call a general instability of the trunk and head. The patient is shaky, and looks as if about to tumble over; his condition is best expressed by the common word "wobble." In addition to this there may be distinct movements not unlike those of chorea. There are deformities; deformity of the spine is one of the commonest, either lateral or angular curvature, or a combination of both; the feet and toes likewise become distorted in a manner which is almost characteristic; the feet become humped and shortened, and the toes exhibit a tendency to curl up. After a time there may be, in addition to ataxia, a certain amount of true loss of power in the lower limbs, accompanied by a certain degree of contracture. Possibly, also, some vaso-motor disturbance sets in in the skin of the lower limb, not unlike what occurs in cases of infantile parapysis.

I will very briefly allude to what is known of the morbid anatomy of the disease. Some twelve or thirteen post-mortem examinations have been made, two of them in this country. In the first place, the nerve-centres are always small, and, in addition to this, there is degeneration running along the white columns of the cord. It appears that the posterior column is principally affected, but not solely, because at the post-mortems, which have hitherto been made in advanced stages of the disease, there is degeneration of the lateral columns as well. So that really there is postero-lateral sclerosis, such as is found in "ataxic paraplegia." I hand you round some plates which

illustrate this fact very well, and a specimen which has been kindly sent me by Dr. Marinesco of Paris.

The great peculiarity of this disease is that it generally runs in families, and for that reason it receives the name "hereditary" ataxia; but "hereditary" does not quite express its common mode of distribution. For usually it is not handed down from parent to child, but breaks out in members of the same generation,—that is, in brothers and sisters; and it may be also in several sets of cousins. True, the handing on from parent or grand-parent to children or grandchildren does sometimes occur, but it is not very common. In one set of cases recorded by Rutimeyer both these features were illustrated. He had under observation four families, in each of which several members suffered from the disease, and he traced these families back to a common great-great-great-grandfather, who was known as "the stumbler." The inference drawn from this nickname was that this ancestor was also ataxic. In the family which I shall presently show you several members are diseased. Their mother tells me that her father—that is, their maternal grandfather—was known to have pains in his legs, and to be unsteady when walking. Here, again, the inference that he had Friedreich's disease is uncertain, for the pains suggest ordinary tabes. The general rule, at any rate, is that the disease breaks out in several members of the same generation, though sometimes, not very commonly, it affects an ancestor as well. At the same time we must take note that sporadic cases sometimes do occur, only one member of a family being affected; then the diagnosis may be somewhat difficult.

As to the further ætiology, it is very difficult to know what it is that really starts the disease in a particular family, and we have not much information on the point. Families in which it occurs are often of a neurotic type, and have generally suffered from some other nervous disease, but that is hardly sufficient to account for such a definite condition as Friedreich's disease. In some cases there is said to have been consanguinity of parents, but I do not think that would hold in the majority of cases. In a large number of instances it has been found that the father has indulged excessively in alcohol. This circumstance possibly, and even probably, may be

regarded as a cause, but the obvious objection to it is that alcoholism in parents is very common, whereas Friedreich's disease is very rare. Doubtless there is more than one cause at work, and when other factors exist, alcoholism or consanguinity may step in and make the difference. I may here allude to some cases recorded by Vizioli, in which this point comes out. He had occasion to observe that a man who had not this disease himself, but who was extremely intemperate, had eight children, who were attacked with Friedreich's disease—an illustration, we may suppose, of the effect of paternal alcoholism. Through one of these ataxic children he had two ataxic grandchildren, which fact illustrates the passing on of the disease from parent to offspring.

The age of onset of this disease is another interesting point in the ætiology. Friedreich's disease comes on at an earlier period in life than the disease with which it is most commonly compared—tabes. Tabes usually occurs after the age of 30, whereas Friedreich's disease attacks the subject before he is 20, the usual age at onset being about puberty. On the other hand, quite young children may manifest the symptoms of the disease as soon as they commence to walk. The probability is that the cord is potentially diseased from birth; that it is small, and is deficient in resisting power, and that actual degeneration steps in as soon as hard work is thrown on the cord in respect to function.

This young woman belongs to a family* whom I have had under my observation for a good many years. There were eight children originally, and she is now one of seven, the other having died young of scarlet fever. Of the seven surviving children, no less than six are affected with this complaint; and, oddly enough, the eldest, a man of 34, is the only one not diseased. This woman is now 21, and has been under my observation eleven years. When I first saw her she could walk, and though a little unsteady, she was not noticeably so, the only apparent symptom she had being loss of the knee-jerk. Then she gradually became more unsteady, and has not been able to walk for the last four years. Her speech is also affected; her feet have a peculiar shape, as you will see, and

her patellar tendon-reflex remains absent. The foot is very much shortened, the arch of the sole, as seen from the inner aspect, is very marked, as seen from the outer very flat; there is a tendency to equino-varus. I think this deformity is partly responsible for the complete inability to walk. A short time back she was in St. Bartholomew's Hospital for an attack probably of influenza, and I then got my colleague, Mr. Bowlby, to do tenotomy. For a little time after that she was able to stand, but the benefit was not permanent. The leg is very cold, and you will observe the bluish aspect of the skin, as if she suffered from chilblains. She has never had any shooting pains, and the sensory condition of her lower limbs is quite normal, except so far as may be accounted for by the chilliness. Her hands also are ataxic, and a little crochet work is the extent of their usefulness. She has a certain amount of curvature of the spine, but not nearly to the same degree as her brother and sister.

The second patient before you is the second eldest in the family, and she has suffered from the disease longer than any of them (sixteen years or more). Nevertheless she is not in a more advanced state in many respects than her sister, who has suffered from the disease eleven years. Notice the extreme arching of the foot and drawing up of the toes, and the tendency to equino-varus. Her legs are not so cold as her sister's, but her knee-jerks are absent. She does occasionally suffer from aching pains in her legs and knees, but not lightning pains. You see that her hands are ataxic, but shutting her eyes does not appreciably aggravate the ataxia. She also has the characteristic speech, a drawing, slurring articulation. The legs and the foot of this patient are markedly affected, and the curvature of her spine has been so great as to make it necessary for her to wear a jacket; this symptom appeared long before she lost the use of her legs. She was under Dr. Sturge at the Royal Free Hospital in 1880 for chorea. She had been knocked down by a drunken woman in the street, and came home trembling, and her hospital paper was headed St. Vitus's dance. Curvature of the spine was discovered shortly afterwards, and subsequently she had a spinal support. You will notice that the curvature is mainly lateral and in the dorsal region; it is not easy to explain its very early

* An account of this family was given by me at a meeting of the Royal Medical and Chirurgical Society, February 24th, 1885.

occurrence. She has a little nystagmus and slight twitching of the forehead, similar to that seen in chorea, and this is interesting in view of the fact that the disease appears to have commenced with a condition indistinguishable from chorea.

The next patient is a member of the same family; he was born next after the girl who has just retired, and before the girl we had in first. His age is twenty-six, and he has had the disease a little longer than his next younger sister. When I first saw him, in 1883, his mother had noticed a "catching" in his legs, and I found that he had no tendon reflex. He was evidently ataxic, but not very much so. He got steadily worse, and his spine became curved, his speech affected, his feet distorted. There is little, if any, nystagmus. Some years ago we had a model taken of his right foot in St. Bartholomew's Hospital, for the museum. Since that time it has altered, and has now assumed the form of equino-varus. You will notice that his feet are bluish red, and they are somewhat cold. His hands also are ataxic, and he is unable to cut up his food, though he can manage to convey it to his mouth. His knee-jerks are quite absent. Like the others, he is now well supported in a chair, but yet you can perceive their general tumble-down appearance; this is partly due to the ataxy of their trunks, and partly to the curvature of the spines. Then the facies is rather peculiar; it might lead one to the supposition that they were half-witted, but I am told that they are sharp and intelligent in every way. In these patients, as in all cases of Friedreich's disease, the pupils are normal, and react to both light and accommodation. In tabes, as you know, the pupil is affected very early in the disease, and reacts to accommodation, but not to light. The spine of this patient you will notice is very much curved in the dorsal region, the curve being chiefly lateral, but partly also antero-posterior. Oddly enough his curvature greatly exceeds that of his elder sister, who has had the disease and the curvature a good deal longer. Curvature of the spine occurs in various forms of nerve diseases in a way we do not quite understand. Friedreich's disease is one such disease, pseudo-hypertrophic paralysis is another, and syringomyelia is a third.

I regret I cannot show you the younger members of this family who can still walk. The youngest one, a girl, I really saw before the disease began,

when her tendon reflex was present. After a time this disappeared, and a year or two after that she began to walk a little unsteadily; so that in this particular case the tendon reflex disappeared before the disease developed.

I now show you a patient from a different stock.* His age is 28, and he has one elder brother who suffers from the disease; it also appears he had another brother who (there seems reason to think) was affected with the disease, but he is now dead. The family consisted of eight; two sisters are alive and healthy. There is no heredity here in the strict sense of the term,—that is, so far as I know no ancestor had had the disease, but I believe there was in the father of this patient the free indulgence in alcohol which I mentioned earlier in the lecture. This man can still walk, though badly, and his gait is a mixed one, characterised partly by staggering, partly by irregular movements of the legs as in tabes. He can manage to stand with his feet together for a little time, but when he shuts his eyes his difficulty is greatly accentuated. His hands are only slightly affected, and he is able to continue his work, which consists of making cases for surgical instruments, and therefore requires some precision. There is no distortion of his feet, but the tendon reflex is quite absent. The plantar reflex is present, and the sensation in the legs is quite normal; there are no pains in the limbs. When trying to touch his nose, his hand shows some unsteadiness, and his speech is similar to that of the preceding cases. He has curvature of the spine, but not nearly so marked as in the last case. The history is that fourteen years ago, *i.e.* when the patient was fourteen years of age, he began to stagger slightly, and gradually got worse until eighteen months ago, at which date he was attacked in the street by a savage dog and upset. Since that time he has been unable to walk alone. I hoped to be able to show you his elder brother, who is rather worse than this man, as he cannot walk at all, though his feet also are quite normal in shape. As regards the brother who died, it is interesting to know that when at school he had St. Vitus's dance, which again raises the question of the relation of chorea to Friedreich's disease.

* Dr. Buzzard kindly transferred this patient to me for the purposes of this lecture. Dr. Sainsbury kindly sent the brother to me. Both patients were shown by Dr. Sainsbury to the Medical Society (January, 1889).

I would now like to say a few words on the differential diagnosis of this disease.

First, the diagnosis from true tabes. This, as a rule, is not a matter of great difficulty. The onset of Friedreich's disease occurs in early life; that of tabes in later life. Family predisposition is a marked feature in Friedreich's disease, whereas it is very rare for tabes to occur in more than one member of any family. Then in tabes there are sensory affections, *e. g.* the characteristic lightning pains and the loss of sensory power in the legs, as well as the Argyll-Robertson pupil, which reacts to accommodation but not to light. Further, in tabes there are a number of what I may call accessory symptoms, such as bladder trouble, a squint, atrophy of the optic nerve, laryngeal and joint symptoms. In Friedreich's disease all these things are conspicuous by their absence. But, on the other hand, in Friedreich's disease there is the peculiar affection of speech, the deformities of the feet and spine, and in later stages the nystagmus, which are absent from cases of tabes.

Another complaint with which Friedreich's disease might be confounded is disseminated sclerosis. I mention this because, when the disease was first described in Germany, certain French authorities held that it was nothing but disseminated sclerosis; they have now come round to the general view. In Friedreich's disease there is the family predisposition and absence of the tendon reflexes, whereas in disseminated sclerosis the tendon reflexes are generally exaggerated. Again, ataxia is rather exceptional in disseminated sclerosis, the more prominent symptoms being paralysis and tremor; the speech affection so common in Friedreich's disease is not so common in disseminated sclerosis, not so frequent as books would lead one to believe. Moreover, the character of the speech differs in the two diseases. In Friedreich's disease the speech is slurring and drawling, while in disseminated sclerosis it is syllabic or staccato, with a tendency to separate each word and syllable. Nystagmus is a symptom which is common to both diseases, but in Friedreich's disease it occurs very late, and may even remain absent, whereas it is often an early sign in disseminated sclerosis.

I have seen Friedreich's disease taken for hysteria, which may be understood if one bears

in mind that both occur in young women, and that the former may occur sporadically *i. e.* in only one member of a family. A young woman whose only complaint is that she cannot stand or walk steadily might easily be put down as hysterical. Still, one symptom should facilitate diagnosis, viz. the condition of the knee-jerk, for this is never absent in pure hysteria, and nearly always absent in Friedreich's disease.

There is one other form of disease with which one ought to draw some contrast, viz. the other form of hereditary ataxia, which I think can hardly be regarded as identical with Friedreich's disease, though doubtless it is related thereto. It has lately been described by Dr. Sanger Brown, of Chicago, and by some other physicians on the Continent. In this, too, ataxy comes on in the legs, then in the arms, followed by an affection of speech, and by choreic movements. But it appears later in life than Friedreich's disease, and is characterised by symptoms which this latter does not possess, such as atrophy of the optic nerve, paralysis of the ocular muscles, and exaggerated tendon reactions. What the pathology of that disease is we hardly yet know. It is supposed by most writers on the subject to be some sort of cerebellar degeneration.

As to treatment of Friedreich's disease, I fear I have nothing to say. I know of no drug or method of treatment which will stop it, and that is what one would expect in a nervous degeneration depending on hereditary taint. I can only say that you must do all you can to keep the health of such patients at the best possible level. It has been noticed that the subjects of Friedreich's disease are particularly susceptible, I will not say to catching infectious diseases, but to the bad effects of such diseases. For instance, a large proportion of Friedreich's own cases died after an attack of typhoid fever. In the cases I have seen infectious diseases seem to bring out symptoms to a marked degree. Of the family I first showed you, one girl attended here for a very slight unsteadiness of walk and twitching of the face, which looked like chronic chorea, and it was only after she had had scarlet fever and recovered that she presented herself with ataxic gait. Another girl got very much worse after an attack of influenza. So that everything possible should be done to prevent patients from getting an infectious disease.

A POST-GRADUATE LECTURE

ON

EXAMINATION OF THE EAR.

Delivered at the London Ear and Throat Hospital, Great Portland Street, May 4th, 1896,

By WILLIAM R. H. STEWART,
F.R.C.S., L.R.C.P., ETC.

Surgeon to the Hospital, and Surgeon to the Ear and Throat Department, Great Northern Central Hospital.

GENTLEMEN,— In cases of deafness, giddiness, noises in the head, and other diseases of the ear, it stands to reason that, in order to arrive at a proper diagnosis, it is as necessary to make a thorough examination as in diseases in other parts of the body. I therefore propose to-night to run through the steps necessary for that purpose, and to explain the various instruments as we go along, afterwards having in some patients for the purpose of demonstration.

A patient consults you for ear trouble, which may include deafness, pain (either inside or outside the ear), a running from the meatus, noises in the head, and giddiness. As usual in such cases, after hearing the patient's own statement it is necessary to cross-examine him, in order to bring out some special points. In conducting this I would suggest a modulation of the tone of voice in which the questions are asked, to get an idea of his power of hearing conversation. Among the questions it will be well to put the following :—The duration of the attack. This is specially important for prognosis, for, naturally, the longer the time the disease has lasted the greater is the likelihood that serious damage has been done to the more delicate structures of the ear. The answer to this question will also enable us to judge of the acuteness or chronicity of the disease. Where intra-cranial complications are present this information also materially aids our diagnosis, for if severe head trouble supervenes within twenty-four to forty-eight hours of the onset of acute middle-ear inflammation, you may be absolutely certain that the disease is that most fatal of all intra-cranial troubles—diffuse meningitis.

It is important to ascertain the mode in which the disease commenced. If suddenly, as in case

of a shock, when the deafness is usually complete and most frequently permanent ; if slowly and with almost imperceptible increase, as in chronic middle-ear catarrh, when the deafness gradually goes from bad to worse. These patients usually say they have been a little hard of hearing for some time, but have lately become much worse ; they are never definite, and a few years more or less seem of small moment, and are much surprised on being told that the loss of hearing has extended over many years, and that very little can be done for them. In such cases our efforts should be directed to retaining what hearing power they have left. The patients never obtain acute hearing again, though some are much improved, and the degenerative process stopped, by a long and continued regular course of treatment. I would particularly emphasize "continued and regular," for if the treatment be not continued with absolute regularity, it does no good at all. Valsalvan inhalations at night of iodine and ether, an alkaline or saline wash for the naso-pharynx in the morning, and an occasional inflation by Politzer's bottle, or through an Eustachian catheter, with a wash out of the tympanum, are of much value. The injection of parolein through the catheter, in some of these cases does great good. These measures should extend over a period of six or twelve months, or even longer, though it is difficult to get patients to properly carry them out on account of the inconvenience they entail.

Next, it is necessary to ascertain whether the ear trouble dated from an illness, for scarlet fever, measles, typhoid, diphtheria, and tubercle are all very provocative of ear disease ; it is, therefore, extremely essential that hygienic precautions be taken during the course of any of those affections, so as to obviate the distressing and often fatal results of middle-ear suppuration which are so frequently seen and which ought never to occur.

General vascular disturbance, such as anaemia and hyperaemia, seriously affect the ear, more especially by producing incessant and unbearable tinnitus.

Injuries and blows must be inquired after, and any obstruction to nasal breathing ascertained. In many instances, however, it is impossible to get at any appreciable cause. The manner and rate of progress of the disease, occupation, place of residence, and family history must all be noted.

With regard to occupation, I would specially mention engine-drivers, who suffer from the quick rush through the air in all weathers, and the continuous noise and oscillation of the engine. Workers in a compressed condition of the atmosphere suffer, not alone from the compression, but also from the relaxation of the air when leaving their work. If such men have suffered from middle-ear catarrh, they ought to wear a small film of cotton-wool in their ears when at work. The same may be said of artillerymen, for their exposure to persistent loud explosions may result in rupture of the drum-head, or the nervous apparatus may be upset if that simple precaution is not observed. These celluloid sound-deadeners which I show you, fit comfortably in the ear and are not manifest.

As to place of residence, damp and insanitary dwellings especially affect the ears. The former produces a tendency to thickening of the nasal and Eustachian mucous membrane, with consequent blockage of the Eustachian tube, or a relaxation of the muscles, more especially the intrinsic and tubal ones, with collapse of the tube ; whilst an escape of sewer gas may have a direct effect on the ear, by producing middle-ear suppuration. Prolonged residence in hot countries may cause a most intense tinnitus and loss of nerve power most difficult to deal with if the patient remains in the same place ; for, as a rule, they only improve or recover when moved to more sanitary and congenial spots.

Deafness being one of those ailments which nature entails as a reminder of parental imperfections, it is obvious that family history has an important bearing on the case. It is a good illustration of the law of atavism—skipping one generation to appear in the next. It usually appears as hereditary taint after puberty. The prognosis is most unfortunate, treatment generally ending in a negative result. Therefore it is of the utmost importance to lessen the chances of deafness as much as possible in all such families by attending to any early indication of approaching mischief, and by being particularly careful to remove enlarged tonsils, adenoids, and any nasal obstructions that may exist.

Inherited syphilis and tubercle, too, are both frequent causes of ear mischief. It is only in cases of syphilitic diseases of the ear that pilocarpine is,

in my opinion, useful. This opinion is based upon repeated trials at the Great Northern Central Hospital and here, both by myself and my colleagues. In non-specific cases patients will sometimes improve under the pilocarpine ; directly, however, the administration of it is stopped, they go back. But specific cases very frequently get absolutely well under the treatment.

Among the symptoms to be noted, pain is perhaps the most important, for it is present, more or less, in all acute inflammatory diseases of the ear. It may be slight, or so intense as to produce delirium, and may radiate all over the side of the head, down the neck, and on to the forehead and jaws. Scarcely any pain is so excruciating as that produced by an inflamed and distended mastoid antrum or tympanum. Acute pains coming on in an old-standing chronic case should be looked upon with the gravest suspicion, for it is in those chronic cases of middle-ear suppuration, in which a fresh acute attack has occurred, that fatal intracranial complications arise. If the pain be of a neuralgic character, and there is nothing visible in the ear to account for it, it is advisable to examine the teeth, as many a carious old stump is responsible not only for pain in the ear, but also for tinnitus. Under all circumstances, pain in the ear should receive prompt attention, so that you may be in a position to at once do battle with any trouble that may arise. In young infants, who of course cannot tell you their trouble, you will find that when in pain they continually put their hands up to the affected part.

Another symptom that requires careful investigation is a discharge from the meatus. The duration of such discharge, the exact spot it comes from, and its character are points which will greatly assist your diagnosis. When first seen the discharge may have lasted only a few hours, as in acute inflammation of the meatus or middle ear ; or, on the other hand, it may have persisted for years, as occurs in some old chronic middle-ear suppurations. The discharge may find its way through one of the fissures of Santorini, the source of mischief being a suppurating parotid. It may come from the cleft in the roof of the meatus, when it may be due to intra-cranial trouble. A spot just behind the tragus may be the site of the exudation, and it may then be caused by a suppurating gland immediately in

front of this prominence. The discharge may, again, take origin in a diffused or circumscribed suppurating inflammation of the soft tissues of the meatus, or from caries of its bony walls. But the most common seat of origin is the cavity of the tympanum or antrum, due to middle-ear suppuration. Inquiry should be made as to whether the discharge is accompanied by pain or tinnitus, or whether those symptoms are relieved by the discharge. Pain and tinnitus in acute middle-ear suppuration are, as a rule, at once relieved when the discharge has burst through the drum-head, or an incision is made to evacuate it ; but if caused by extensive caries the discharge will continue great, even after the rupture has taken place. A thick, creamy, sweet discharge points, as a rule, to recent trouble, and is the more easily cured ; but if foul, thin, and perhaps tinged with blood, it indicates long-standing mischief, with either dead bone or an antrum filled with inspissated pus, the stench of which is at times simply abominable. In these latter cases, where the discharge is merely the overflow from a distended antrum, I cannot too strongly urge the immediate opening of that cavity and the establishment of free drainage after thoroughly douching and scraping. The pus at times clings to the walls with such tenacity as to require a more or less free application of a sharp spoon to dislodge it. The wound should not be allowed to close until the bone is in a healthy condition. Under this treatment the patient soon gets well ; otherwise the discharge goes on for years, defying all endeavours to stop or sweeten it, the patient all the time incurring the greatest risk of losing his life from intra-cranial complications. Never leave a case of otorrhœa until it is cured if you can help it, and if you have anything to do with insurance companies never pass a life as good if a discharge exists.

Tinnitus and vertigo are also important symptoms. On the one hand they may mean that only a very small piece of cerumen or dust is on the drum-head (the smallest speck at times causes most distressing symptoms), or it may be due to a slight disturbance of the general system, such as the liver being out of order. On the other hand, those symptoms may indicate most serious and intractable ear disease, or sound the alarm of grave trouble in more remote regions, such as the kid-

neys, heart, liver, or uterus. Some of the most intractable cases of tinnitus arise at the menopause.

I will now show you the instruments which are generally used, and I divide them into four sets—

1. Those required to make a direct examination.
2. Those required for ascertaining the hearing power and condition of the auditory nerve.
3. Those for inflating the tympanum and ascertaining its condition.
4. Those required for clearing out the external meatus and applying remedies.

Under the first head, the chief requirement is a bright and steady light. If obtainable, sunlight is the best ; both the oxy-hydrogen and the electric light are good, but the oxy-hydrogen is rather too strong, and its continuous use may affect the eyes.

The electric light is perfect : too large a lamp should not be employed ; that which I am in the habit of using is 32-candle power, and is sufficiently strong for any purpose. In default of either of these, an ordinary bull's-eye with an Argand burner and racket bracket, or even a Queen's reading lamp, are of service. One great objection to these is that when patients are required to sit close to them for any length of time they complain of the heat. However, they can now be made with a double chimney, the inside of which is sometimes lined with asbestos, which keeps away the heat. If none of these sources of light are to hand, a candle, or even a wax taper may be used.

Next, a refracting frontal mirror is required ; the one I prefer is Mackenzie's, with spectacle frame. Some use a hand mirror, but in that case there is the disadvantage that one hand is kept occupied.

A set of aural specula are needed ; I prefer Gruber's, a set of four conical ones. Kramer's, which is capable of being expanded, is useful when operating or applying remedies. I have a small one of my own, which consists of small Kramer-blades on a stout spring like Thudichum's nasal speculum, which keeps it in place, giving the operator the use of both hands. But the spring must only be just strong enough to hold the speculum in the ear, or pain will be produced if inflammation exists. Some prefer John Brunton's, which I show you ; with this a frontal mirror is not required, but it is useless if you have to do anything in the shape of an operation. I do not like it

because it magnifies too much. Seigel's speculum—a vulcanite speculum which screws into a vulcanite box with a glass end ; an india-rubber tube with a mouthpiece is connected with the box, to which suction can be applied ; a piece of rubber tubing on the speculum portion makes it fit air-tight in the meatus—is necessary for ascertaining the mobility of the drum-head, as well as for applying a certain amount of massage, viz. by alternately exhausting and filling the meatus.

As in the large majority of cases of ear trouble, the mischief starts from the naso-pharynx ; it is absolutely necessary to first examine the pharynx and naso-pharynx, as well as the anterior nares. For the anterior nares this modification of Thudichum's speculum is the best. For posterior rhinoscopy, Zauful's posterior rhinoscopic mirror, which works on a hinge, or an ordinary small mirror bent at an angle, will answer every purpose. A tongue depressor is also needed ; it should be well roughened at the end, and fairly broad, to enable the tongue to be drawn well forward.

For ascertaining the hearing power, and the condition of the auditory nerve, a watch and a tuning-fork are really the only instruments which are absolutely necessary for ordinary work, and the manner of using these I will explain to you when the patients come in. For inflating the tympanum and ascertaining its condition, a Politzer's bottle is needed. It consists of an india-rubber bottle holding six to eight ounces, with a valve at the bottom, and conical rubber nozzles. I prefer these nozzles, because if the patient jerks forward during inflation no damage is done to the mucous membrane of the nose. Politzer's inflation consists in directing the patient to hold some water in the mouth ; the nozzle of the bottle is then inserted into one nostril, the other being closed by the index finger and thumb ; the patient is then told to swallow, and at the end of the second stage of swallowing, when the nasal cavity and upper part of the pharynx are closed by the soft palate the bag is compressed, and air blown into the tympanum. This is the true Politzer's method ; the best substitute is directing the patient to close the lips and make a forcible expiratory effort. This saves the trouble that sometimes occurs when the patient, not doing what he is told, the water is blown into the larynx, etc. ; but strange

as it may seem, some patients cannot blow out their cheeks, in these cases water must be used. The tympanum can also be inflated by means of the Eustachian catheter ; we generally use the short ones I show you, about four inches in length. We have them made of both silver and vulcanite. I prefer the silver ones, because they may be bent as far as required, and may be immersed in boiling water immediately after use. A single hand-ball bellows is necessary, to which a loop should be attached to hang on a button of the coat to leave the hands free ; it should also have a nozzle made to fit the catheter. An auscultation tube is a piece of rubber tubing with two nozzles, differently coloured, so that one may be used for the ear of the examiner and the other for that of the patient. The method of inflating is as follows :

Having hung on the hand-ball bellows, the auscultation tube is inserted, the catheter is then passed in the following manner. The instrument is held parallel to the plane of the face, and the beak inserted ; the hand is gently and quickly raised until the instrument is at a right angle with the face, the beak having been at the same time pushed along the floor of the meatus ; if the hand will not come up to the right angle, you may be pretty sure that the beak has slipped out of the inferior meatus, it is therefore as well to withdraw and begin again. When the beak touches the posterior wall of the naso-pharynx, the instrument should be turned round until the ring points to the outer canthus of the eye, then by gently pressing towards the middle line and slightly withdrawing, the beak will be felt to slip over the posterior lip of the Eustachian tube, then slight pressure places it in the mouth of the tube. The nozzle of the bellows is then inserted into the catheter, and the ball gently squeezed. I would here warn you that on using any of the instruments I have mentioned, or in any manipulation or operations on the ear, be careful to avoid all force. No force is ever necessary, and much damage may be done if it is used, not only to the patient but to your own reputation. The sounds heard through the auscultation tube when inflating are the following : if the passage is free from obstruction, you will have a full clear sound as the air strikes the drum-head. If an obstruction be present, the sound will be modified according to the degree of obstruction. If there

be a perforation, you will hear a distinct whistle right in your own ear. If fluid be present there will be a gurgling sound; while in a dry, sclerosed middle ear a crackling sound is perceptible. If you wish to inject a fluid into the tympanic cavity, after having first ascertained that the catheter is in the mouth of the tube, the nozzle of the bellows should be withdrawn, the fluid taken up with a pipette and dropped into the catheter, the nozzle is then replaced and the fluid gently blown up.

Patients may inflate with a particular solution at home by putting the fluid, which should be only just hot enough for the steam to come off, into a jug with a narrow mouth (which should be put into a vessel of hot water to keep the temperature uniform), then with the hands around their mouth and nose draw the steam in through the mouth and nose; these are then closed and an effort at expiration made, the steam is thus driven through the Eustachian tubes into the tympanum. Only sufficient effort should be made to gently inflate the tympanum, as evidenced by the sensation of cracking in the ears and the feeling of something warm in them. By keeping the mouth and nose closed, and swallowing, the tympana are emptied. Patients must be warned against the too frequent or forcible use of this Valsalva method, or the drum-head will get relaxed. Another warning I would give is to exercise extreme care with patients who suffer from cardiac disease, as syncope may follow. A lady, too, once came to me when she was six months pregnant. I used Politzer, and she went home and miscarried. This may be only a coincidence, but it is worth while remembering.

Among the instruments for cleaning out the external meatus and applying remedies, the most useful and most dangerous is the syringe. I use a brass one holding three or four ounces, such as this. The water or fluid used should always be warm, and it is well to let the first shock strike the inside of the concha, and remembering the shape of the meatus, straighten it as much as possible by drawing the ear backwards, outward and upwards, and direct the stream of water along the roof. Never syringe an ear until you have examined it and satisfied yourself that there is something to syringe out. Do not syringe for too long a time without an examination, and recollect that vertigo, sickness and syncope may

occur. When cerumen is present it is as well if the plug is inspissated, to attempt to soften it by inserting a warm solution of bicarbonate of soda for two or three nights. Do not use instruments to remove an obstruction until you have tried over and over again and absolutely failed with the syringe. If instruments be required to remove foreign bodies or small polypi, the angular forceps I show you are very useful. Polypi, if not very large, come away better with this than with a snare, as a snare may slip and leave a bit of the growth behind. A ring-knife is required to remove small granulations, as well as to get rid of débris in the ear; so also is a probe, and all these instruments should have handles at an angle so as not to get in the line of vision. For cotton-wool holders I always use thin iron, roughened at the end. When chromic acid is used to destroy granulations, a carrier is wanted; it has a platinum point in which is a fine slit for holding the fluid. A platinum crucible must also be included among your instruments for fusing nitrate of silver. Snares are especially necessary for nasal polypi, and there are many varieties of the instrument; the best ones are those in which the handle keeps out of the range of vision, and have both pulling and screw power. For operating on post-nasal growths I always use Gottstein's ring-knife, which has one great advantage over the old forceps,—namely, economy of time, as it enables the operation to be done in about thirty-six seconds, so that gas alone need be employed. If forceps are used the finger should first be inserted; I have seen the mucous membrane covering the vomer and a portion of the bone itself removed in the absence of this precaution. There is a case on record, too, in which, in tearing out a growth, I presume with blunt forceps, a vein was torn; there was a gush of venous blood, and the patient died on the table. For removing the tonsils there is nothing so good as a Mackenzie guillotine, because it is practically impossible to do any harm with it. The turbinate bones and spurs from the septum are, I think, interfered with much oftener than they ought to be; but when their removal is absolutely necessary no instrument answers the purpose so well as Carmalt Jones' spokeshave. For operations about the nose and for aural polypi, cocaine is required. I always employ a plug of

cotton-wool soaked in cocaine and placed upon the spot. For throat work I prefer a brush, which is more satisfactory than a spray, for by the latter means one cannot tell how much gets into the stomach. A case is recorded of a patient having died from cocaine poisoning after the use of the spray. The further instruments necessary are a cautery battery, a knife for perforating the tympanic membrane, and the various gouges, scoops and trephines for mastoid disease and intracranial complications; these latter I shall explain more fully to you at my next lecture. Schall makes a very nice battery of four cells, which does not get out of order if properly looked after.

[The lecturer then reviewed the anatomy of the ear and adjacent parts, and demonstrated the use of some of the instruments on patients.]

THERAPEUTICAL NOTES, &c.

The Treatment of Soft Chancre.—The following treatment is recommended. Every morning the ulceration should be touched with a tampon of cotton wet in this solution :

Menthol	gr.j
Carbolic acid	gr.v
Alcohol	f 3iss

Aristol is next dusted on, and a piece of absorbent cotton applied, thus completing the dressing. The patient is directed to wash the sore several times with carbolic acid solution, and to dust with aristol. All friction or causes of congestion are to be avoided.

Phagedenic ulcer is touched with solution made up of :

Cocaine	gr.j
Potassio-tartrate of iron	gr.xv
Distilled water	f 3iss

It is next powdered with a mixture of 20 parts iodoform, and 5 parts menthol; and potassio-tartrate of iron is given internally.

(*La Tribune Médicale.*)

Treatment of Perforating Ulcer by Nerve Stretching.—Dr. Chipault reports five cases of perforating ulcer successfully treated by stretching of the plantar nerves. After many unsatisfactory attempts to deal with this affection by amputation, incision of the ulcer, and scraping, the idea

occurred to the author that stretching of the nerves distributed to the region occupied by the perforating ulcer might give some good results. Rejecting cases of non-trophic perforating ulcer, which may be regarded as unamenable to this plan of treatment, he tried nerve stretching with an unexpected degree of success in two cases in which the perforating ulcer was due to peripheral neuritis, and in three others of spinal origin. The five patients, all of whom had suffered for many years and undergone useless amputation, were in the course of a few days after stretching of the plantar nerves relieved of the ulceration and of the concomitant trophic disturbances. As four patients were kept in bed for only twenty-four hours after the operation, and the fifth was thus confined for eight days, these good results could not be attributed to the rest which so often effects a transitory improvement in cases of perforating ulcer. These patients have remained quite free from their affection after intervals from the date of operation of eight, seven and a half, seven, six, and three months. The author has practised stretching twice of both the internal and external plantar nerves, once of the internal plantar alone, and twice of the internal collateral nerve of the great toe.

(*La Presse Médicale.*)

Incontinence of Urine.—Dr. M. J. Stumpf employs the following method with favourable results. During sleep raise the pelvis of the child so as to form an angle of from 130° to 140° with the vertebral column. This position offers an obstruction to the passage of the urine into the urethra, and the sphincter is not excited. Dr. Stumpf has cured twelve children and one adult in this way. After three weeks of treatment there need be no fear of recurrence, and the child may be allowed to sleep in the normal position.

(*Gazette des Hôpitaux.*)

Theory to Account for Melæna.—Loranchet attributes melæna to the gradual chilling of the surface of the body after birth. The cold acts as a general debilitant and depressant of the nervous system; general circulation is disturbed; peripheral circulation is slowed; the vaso-motor system is unbalanced. The change from the splanchnic cycle of the foetus to the cardio-pulmonary cycle of the new-born infant is interfered with, and in the struggle of the reflexes there is a reversion to the splanchnic cycle with passive congestion of the gastro-intestinal mucous membrane, which gives rise to the haemorrhage.

(*Gazette Heb. de Méd. et de Chir.*)

THE CLINICAL JOURNAL.

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A CLINICAL LECTURE
ON
MITRAL STENOSIS.
BY
S. H. HABERSHON, M.D., F.R.C.P.

GENTLEMEN,—In order to grasp the meaning of the various abnormal cardiac sounds that we hear in mitral stenosis, it is most important to bear in mind the mechanical events in the cardiac cycle and the periods at which they occur. The heart, as our physiological text-books teach us, partakes of the twofold character of a force-pump and a suction-pump. The force-pump action takes place during the systole when a wave of contraction passes from base to apex, the contraction of the auricles being succeeded almost immediately by the ventricular contraction, the two sides of the heart acting simultaneously. The blood is then propelled in the direction of least resistance by the sudden narrowing and contraction of the cavities in turn. The cycle commences by the auricular systole or contraction, the onward rush of blood passing through the auriculo-ventricular orifices, and widely reflecting the cusps of the mitral and tricuspid valves. If the orifice is roughened or constricted a murmur is produced, known as the auricular systolic or the presystolic murmur.

The tension in the auricles necessarily rises rapidly during their systole, reaching its maximum as the ventricular systole commences. The already increasing tension in the ventricles receives a sudden reinforcement when their contraction commences, rising, as in the case of the auricles, to a maximum at the close of the ventricular systole. During this period blood is forced through the aorta, and partly by the contraction of the musculi papillares and partly by the reflux of blood the auriculo-ventricular valves are floated up and completely close the backward exit. Thus the events taking place during this period of the cardiac cycle

are the contraction of the ventricles, the closure of the mitral and tricuspid valves as soon as contraction commences, and the onward rush of blood into the aorta and pulmonary artery. All are systolic in time, and include the period of the first sound which marks the beginning of the period.

The aorta and pulmonary artery are already full, and dilate to receive the blood forced into them at high pressure by the ventricles. Their distension or dilatation is followed by a rebound producing a reflux of blood, which again closes the backward outlet by floating up the cusps of the sigmoid valves, closes them sharply, and produces the second sound of the heart. Whilst this onward rush of blood is taking place through the great arteries, the auricles first, and next the ventricles, are commencing to relax, and blood is slowly entering from the pulmonary and systemic veins. The ventricles dilate more quickly than they can be filled, and hence pressure falls and reaches its lowest point immediately before the closure of the sigmoid valves. At this period the pressure in both ventricles is negative. The dilating action of the ventricles is not a passive action, but is comparable to the sudden expansion of an elastic bag after it has been compressed. This elastic reaction produces a powerful suction action, assisting the passage of blood through the auriculo-ventricular orifice. Necessarily the suction action is greatest at the period of lowest tension, and this coincides (as the endocardial pressure tracings of Marey demonstrate) with the closure of the semi-lunar valves. The suction action, however, continues as long as the ventricles dilate, until in fact the dilatation gives place to the next ventricular systole. Authorities differ as to the period of greatest suction. Some writers, such as Jaccoub, and lately Rolleston, place the greatest effect of suction at the commencement of the relaxation of the ventricles, at about the period of the second sound, continuing more or less during the whole long pause until the succeeding cardiac cycle commences. Others, as Acland, prefer to consider the suction-pump action greatest at the end of the relaxation of the ventricles during the

period of the auricular systole, thus aiding by a *vis a fronte* the *vis a tergo* action produced by the contraction of the auricles. I prefer to consider the action as greatest at the period when tension is lowest, and therefore when any relaxation would tend to produce a still further diminution if it were not for the entrance of blood from the auricles to fill the vacuum. It is obvious that under some conditions, and chiefly when the orifice through which blood is sucked is roughened or constricted, that a murmur diastolic in time may be produced.

This is, in fact, the chief reason for the diastolic murmur that is heard in some cases of mitral constriction, and of mitral regurgitation in young persons. The murmur may either replace the second sound or obscure it at the apex, in which case it is known as the early diastolic murmur of mitral disease; or it may occur during some part of the long pause, when it is described as the mid or late diastolic murmur. Dr. Acland considers that the roughness of the presystolic murmur is largely due to the suction of the ventricle, reinforced by the auricular contraction in cases of mitral stenosis.

So far we have considered the heart as an organ governed mainly by mechanical conditions. Though these conditions play an essential part in the causation of both physical signs and symptoms of valvular disease, we must not lose sight of the fact that the heart is a vital organ, subject, as other tissues are, to the general laws of nutrition and to great variations in the condition of its walls, the tension of the blood in its cavities, and the nervous mechanisms which control its action.

(a) The condition of the cardiac muscle is capable of great variations both in its contractile power and elastic expansion, this alteration being dependent in great measure upon the nutrition of the cardiac walls, the resistance against which its power is exerted, and the inflammatory or degenerative changes which may supervene.

(b) The tension of blood filling the cavities is also a variable quantity. It is influenced by vaso-motor changes in the systemic arterioles, by alteration or diseases of their walls, by the respiratory movements and the condition of the pulmonary circulation, and by peripheral obstruction due to disease.

(c) The heart possesses a nervous mechanism which initiates the rhythmic movements of the

organ, controls and co-ordinates the various actions, and is itself capable of either depressing or exciting the cardiac muscular movements when stimulated by the reflex influences conveyed to it by means of the vagus from the cardio-inhibitory centre. This centre may be influenced by stimuli proceeding from many parts, e.g. liver, abdominal organs, &c.

All these variable influences convert a simple mechanism governed by ordinary physical laws into a complex organ, the complexity being due to the difficulty of estimating which varying factor is in excess.

The physical and mechanical laws, then, which affect the circulation and the heart in particular, are tempered by vital changes in the tissues of the heart. Many of these changes we include under the name of "compensation." I would here remind you of the general law enunciated by Sir James Paget, which, as one author (I think Gowers) points out, has a special reference to the walls of cardiac cavities, viz. that obstruction to the outlet of fluid from any cavity in the body produces dilatation and hypertrophy of the walls of the passages behind the obstruction.

This applies also to the case of regurgitation through a valve, which may be regarded as an obstruction to the onward flow of blood. In mitral regurgitation, though the law holds for cavities behind the mitral valve, there is this exception, that the cavity in front of the valve (the left ventricle) also tends to dilate and become hypertrophied, and aortic dilatation is not an unfrequent result of regurgitation through the aortic valves.

Mitral Stenosis.—The dangers of mitral stenosis lie in the fact that it is a progressive disease. The valves, originally puckered and roughened by endocardial inflammation in the earlier stages, frequently produce an incompetence as well as an obstruction. But it is the tendency of the mitral curtains to become further glued together and thickened, not only by repeated inflammatory attacks, but by an adhesive inflammation of an exceedingly chronic character. This is thought to be set up partly by the irritation due to friction and the intermittent contact of irregular and roughened surfaces, and partly by the increased strain to which they are subjected by the heightened tension induced behind the obstruction. The *button-hole* and the funnel-shaped openings that are often seen on the post-mortem table in an advanced

case of mitral obstruction, have been thus produced.

In cases of pure mitral stenosis without regurgitation the obstruction has its effect only on the cavities behind the mitral valve, and no assistance is to be obtained from the left ventricle as in the case of mitral regurgitation, because the tendency is (at all events if the obstruction is extreme) for less blood than usual to enter it, and with a low tension and an imperfectly filled cavity the left ventricle not only does not become successively dilated and hypertrophied, but is often found to be atrophied. This atrophy is only observed, however, in extreme cases, and, as I have said, in early cases we frequently obtain the combined result of regurgitation as well as obstruction.

The important difference, then, between *mitral narrowing* and *mitral regurgitation* is that in the former the obstruction behind tends constantly to increase, and there is no safety-valve action obtained in the compensating power of the left ventricle when the block becomes extreme.

As in the case of mitral regurgitation, the tension is transferred backwards, and we find enlargement of the left auricle, the right ventricle and auricle, and increased pressure in the lungs; and remembering that the tension is always tending to become increased more surely and progressively than in mitral regurgitation, we find the physical signs of an increased tension almost more constant.

Thus in the earlier stage of the disease we note a distinct evidence of hypertrophy of the left auricle. The auricle becomes dilated, and the auricular appendix approaches the surface of the chest in the third left intercostal space, and its conducted pulsations can often be seen. The second pulmonary sound is almost invariably accentuated, indicating the heightened tension in the pulmonary artery. In advanced disease the right ventricle becomes dilated and hypertrophied, and may altogether displace the left ventricle, and be found beating far to the left of the normal apex.

Mitral stenosis has led to much controversy on account of the peculiarity of the murmur which accompanies it, and which indeed is acknowledged by all observers (whatever their view of the mechanical cause that leads to its production) to be pathognomonic of the affection.

The so-called presystolic murmur occurs at the end of the diastole, immediately preceding the first

sound of the heart. It is often extremely rough and grating, and leads up to the first sound, which is unusually short and sharp. It is commonly but not invariably accompanied by a thrill. I shall follow the description of the disease given by Jaccoud, and later in the excellent treatise of Broadbent, who divide the disease into three stages.

In the *first stage* the constriction is not extreme. The *murmur is loud and rough*, ending in a sharp first sound, and the second sound is plainly heard at the apex, which is little if at all displaced to the left. In this stage, as Broadbent states, the murmur is so characteristic that the diagnosis is easy. There is no great evidence of dilatation of cavities, though there may be slight extension of dulness upwards to the left of the sternum. The *second pulmonary sound is often accentuated*. The heart usually beats regularly, though sometimes with greater frequency than usual. The *pulse is regular* and not particularly small.

The *second stage* is characterised by the *starvation of the left ventricle*. With increasing constriction the tension in the ventricle falls, because it is imperfectly filled. There is not sufficient tension to close the aortic valves vigorously, and therefore the *second aortic sound* heard over the aorta is feeble, and is diminished or even absent at the apex of the heart. Broadbent states that an additional cause for its absence is that at the apex the enlarged right ventricle displaces the left. At the same time the first sound becomes *sharper* and more of the *character of a snap* (as Acland has described it) than before. This is due to several causes, partly the irritability of an imperfectly nourished ventricle, and the so-called irritable beat of the heart is often caused by a weak ventricle, and (Broadbent explains*) to the fact that there is less resistance than usual to the contraction of the ventricle, and it contracts more rapidly in consequence, and is suddenly made tense when it meets with resistance, at the same time bringing together the rigid valves with a sudden shock.

Besides this alteration in the character of the sounds, the second sound, except at the apex, is frequently reduplicated. This, as well as an accentuation of the second pulmonary sound, is an indication of an increase of tension in the pul-

* "American Journal of Medical Sciences," No. 68, 1886.

monary artery and a more hurried closure of its semilunar valve. In this stage the cavities, especially the left auricle and right ventricle, become dilated and hypertrophied, and all the evidences of an embarrassed circulation are seen. The heart becomes enlarged in all directions, and the apex is displaced by the increase of size in the right ventricle. The *presystolic murmur* is still heard, but often becomes *extremely prolonged*. Broadbent states that it may occupy the whole diastolic interval between the second and first sounds, or this murmur may be divided, and a murmur be heard at or immediately following the second sound of the heart, and after a short interval the ordinary presystolic murmur closing the diastole and ushering in the sharp first sound.

The *prolongation of the presystolic murmur* is probably brought about by the increasing hypertrophy of and more prolonged contraction of the left auricle, just as we find that when the left ventricle is hypertrophied the first sound of the heart is lengthened. The murmur occurring at the commencement of diastole, at or about the period of the second sound, and frequently fusing with the prolonged presystolic murmur is due to a different cause. According to its relation to the second cardiac sound it has been called the early or mid-diastolic murmur. It is heard at the apex of the heart, and like the presystolic murmur is not heard behind, thus pointing to its conduction in the onward direction of the blood-stream. It must be distinguished from the murmur of aortic regurgitation, which indeed is a common cause of a diastolic murmur heard at the apex of the heart, for aortic disease is not an infrequent accompaniment of mitral stenosis. It is therefore not heard at the base of the heart.

We must look for its explanation in the mechanics of the cardiac circulation, and I must ask you to go back for a moment to my description of the events taking place during the diastole of the heart. After the contraction of the ventricles there is a sudden pause, during which the rebound of the aortic walls in response to the sudden influx of blood closes the aortic valves. At this point the tension in the ventricles is at its lowest. The ventricular walls suddenly relax, and their expansion is not passive, but is of the nature of an active elastic reaction. A powerful suction is produced, and blood enters the cavity from the auricles.

When the tension in the left auricle is increased, the force of the onward rush of blood is greater than normal; and if the orifice is roughened or contracted, as in these cases of mitral constriction, a murmur is produced, but more frequently at the commencement of the ventricular relaxation, when the suction power, aided by the heightened auricular tension, is greatest. Dr. Gowers, in an article on Dilatation of the Heart ('Reynolds' System of Medicine,') suggests an additional reason why the diastolic murmur frequently ceases before the commencement of the presystolic murmur. He says "this silence can only be explained by a cessation or almost cessation of the flow of blood, which means, of course, *an equalisation of the pressure in the two cavities.*" I believe this to be the cause of the diastolic murmur which I have heard in cases of mitral regurgitation in children or young persons with a greatly hypertrophied heart and a dilated ventricle, but it presupposes a roughening of the mitral orifice. A murmur caused by blood flowing through a narrow orifice can only be produced when the velocity of the stream is sufficiently great; and in these cases of mitral constriction with a presystolic and diastolic murmur, it is produced at the two periods in the cardiac cycle when the blood is flowing most rapidly,—first, when suction commences, and the high tension in the auricle hurries the stream through; and secondly, when the weakening current is again reinforced by the auricular contraction.

The third stage of mitral stenosis is that of increased constriction, general failure of compensation, and grave results of an obstructed circulation.

The characteristic presystolic murmur disappears on account of the *giving way*, that is, *the dilatation of the right ventricle*. This relieves the pulmonary tension, and, since there are no valves in the veins, also the *left auricle*. In extreme cases the second pulmonary sound is no longer sharp, and the tension in the left auricle is not sufficient to produce a murmur at the mitral orifice. The dilatation of the right ventricle at this stage frequently gives rise to a systolic murmur heard at the ensiform cartilage, and along the left border of the lower end of the sternum. This murmur is due to tricuspid regurgitation, and is accompanied by other signs, pulsation in the jugulars, and general venous engorgement, and sometimes pulsation of the liver.

Though the auricular tension is now not sufficiently great to produce a presystolic murmur, still the left ventricle is not properly filled, and the contraction retains its sharp and sudden character, while the second aortic sound is lost because the aortic tension is low and produces a feeble reflux.

At the apex Broadbent states that frequently nothing is heard but a *sharp clicking sound*. The right ventricle becomes extremely dilated, and enlarges the dulness of the heart to the right. It may also occupy the position of the true apex, and it is not uncommon for the systolic murmur to be mistaken for that of mitral regurgitation, because it is heard at the apex of the heart. The difficulty is still further increased by the fact that the second sound, which is the second pulmonary sound, is heard over the right ventricle. As a rule, however, the sharp clicking first sound can be heard through the systolic murmur, and the second sound over the aorta is weakened. The diagnosis in such cases is, however, one of great difficulty.

The dilatation of the right heart leads both to a want of synchronism in the ventricular contraction at the two sides of the heart and to an irregularity and rapidity of beat. It is not uncommon to hear a cantering action of the heart (doubling of the first sound); while at times the heart's action is tumultuous, and a confusion of sounds is heard. Broadbent points out that the very difficulty of deciding upon the explanation of the sounds is often a proof of the existence of mitral stenosis. An *important condition of pulse is seen at this stage*, that is in cases where there is great constriction. The contractions of the left ventricle are often so feeble that they are not conveyed to the pulse, and hence the radial pulse-beats are found to be much fewer than the number of cardiac contractions, and of extreme feebleness. The heart-beats may be even twice as numerous as the pulse-rate. Moreover the heart may be regularly irregular, and the alternate beats, or every third beat, may be stronger, thus producing an alternating or duplicating pulse.

Mitral stenosis, as has been previously remarked, is frequently accompanied by regurgitation with or without a systolic murmur, and this further complicates the physical signs because the left ventricle may then become dilated and hypertrophied. Such a combination produces what is often called

the round heart of mitral disease, with an area of dulness extremely extended to the left and to the right.

Several examples will be shown you in which the aortic valves are also affected. This is a very common complication, and there is no doubt that the combination of aortic regurgitation with mitral stenosis, adding as it does to the obstruction at the aortic orifice, greatly increases the gravity of the disease (Balfour). From what has gone before it will be seen that the diagnosis of mitral obstruction, except in the early stages and in the presence of the characteristic recurrence, is not easy.

In the later stages care must be taken to observe if the first sound is sharp and clicking, and if accompanied or obscured by a systolic murmur. The reappearance of the presystolic murmur may often be observed when the patient is placed at rest. The character of second sounds at the base should be noted and compared, and the absence of the second sound at the apex. The want of correspondence of the heart and pulse beats with the smallness of the radial pulse and the presence of a diastolic apex murmur, without aortic regurgitation and a jerking pulse, give additional aid to the diagnosis.

In the diagnosis of mitral stenosis there are several practical points to be observed:—

First. Never forget to examine your patient in the recumbent posture. All mitral murmurs are increased in this position, and often, when the sharpened character of the first sound in the erect position leads to the suspicion of mitral stenosis, no true presystolic murmur is heard until the patient lies down. The presence of the presystolic murmur is associated with a certain amount of vigour in contraction. A patient may be admitted to the hospital without the characteristic murmur, but after a few days in bed, and when the rest has to some extent increased the power of the heart, the murmur will return, and this is in itself a favourable sign.

Second. The size of the right ventricle. As I have said, it is often the case in advanced disease that the size of the right ventricle becomes very great. It displaces the true apex of the heart formed by the left ventricle, so that the apex beat is now formed by the surface of the right ventricle. If in addition there is extreme dilatation and a tricuspid systolic murmur, this murmur may be

heard all over the cardiac area, and be easily mistaken for the systolic murmur of mitral regurgitation. I have seen this happen. How then in these cases can you distinguish between a dilated right ventricle and a dilated left ventricle? By the character and position of the second sound. It must be remembered that the second sound, heard at the apex of the heart (*i.e.* over the apex of the left ventricle), is the second aortic sound; and that heard over the right ventricle is the second pulmonary sound. This can easily be corroborated by comparing the sounds heard in these situations with the second sound at the base of the heart.

In an extreme case of mitral stenosis at the true apex there is no second sound heard, or it is very feeble, and we may corroborate that by placing our stethoscope over the second right intercostal space, and there we shall find the aortic sound very feeble. If we pass from what we imagine to be the true apex along the line of the fifth interspace, and listen over the front of the heart, and if the right ventricle is occupying that area, we shall now hear a very distinct and perhaps sharp second sound, and on listening in the pulmonary area we shall find that the second pulmonary sound is also accentuated. If, then, there is a systolic murmur all over the heart, and the second sound accentuated, but at the aortic orifice you find it feeble, you may be sure you are listening over the right ventricle and you are listening to a murmur of tricuspid regurgitation produced in the right ventricle. I shall be able to illustrate this point more lucidly in the case of some of the patients who are here this afternoon.

The various stages of mitral disease are not marked with the precision that has perhaps been conveyed by the description I have given. A case of moderately advanced disease may come to the hospital with sudden and extreme dilatation of the cavities behind the obstruction, and many of the cases we have to treat in the out-patient room come because of this temporary and sudden condition.

A NOTE FROM THE CLINIC OF

C. B. LOCKWOOD, F.R.C.S.,

Assistant Surgeon to St. Bartholomew's Hospital, Surgeon to the Great Northern Hospital, etc., etc.

An Unsuitable Case for the Operation of Radical Cure of Hernia.

THIS case, which has been sent to me to have the operation of radical cure done, is a good example of the pitfalls which beset that operation. When done upon the right kind of cases its results are perfect, and surgery has not a better instance; but done upon unsuitable cases a relapse is sure to occur, and the operation be brought into disrepute. The suitable cases, those in which you can promise a cure after an *efficient* operation, are children, young adults, young men and women with congenital hernia. In these, as I have shown elsewhere, the rupture is due to a developmental defect in the wall of the abdomen. There is nothing wrong with the tissues themselves, and all the suspensory apparatus of the mesentery, intestines, and abdominal viscera is normal. Clearly if the aperture in the wall of the abdomen can be efficiently closed, the intestines or omentum will cease to escape. But it is always necessary to remember that no one can deprive human beings of their liability to hernia; unfortunately, all are so constituted, that under certain circumstances the intestines or omentum may escape from the abdomen.

If we proceed with the case before us point by point, it will be easy to show why it is unsuitable for operation. The man says that he is a plate-layer, and that he is forty-eight years of age. First let us look at the whole patient. It is easy to see that, like most of those who have followed a laborious occupation, he looks worn and has lost his youthful vigour and elasticity. Indeed, in most London working men at forty-eight years of age the tissues have begun to deteriorate and lose their tone. Presently we shall see that in his case this has led to some relaxation of the abdominal wall, and probably to some prolapse of the mesentery. Obviously a person with a bad abdominal wall and a prolapsed mesentery is more predisposed to rupture than one whose tissues are perfect and whose intestines have the usual limited range of movement.

Given these conditions, his employment must have an influence upon the occurrence of the hernia, and also upon the liability of the hernia to relapse should anyone perform an operation. I can imagine that even in bad cases of acquired hernia the abdominal wall can be made strong enough to support slight exertions, or if the patient's occupation is very light. But a plate-layer has heavy weights to lift, and I doubt whether any operation could afford him more than a short respite from hernia. Three months after the operation suitable cases can do anything they like. Some of those upon whom I have operated have hunted, shot, played football, ridden upon horse-back, ridden upon gun-carriages, made boilers, and so forth, without any trace of relapse. They are cured of their ruptures, but it is necessary to remember that they are not deprived of the predisposition to rupture which every human being possesses. They may, just like anyone else, rupture themselves again by efforts beyond their strength.

The history of this man, as usual, tells us very little. He asserts that his rupture came suddenly in the right side whilst he was lifting timber. Congenital herniae often come suddenly under similar circumstances, but they are usually very painful and often strangulated from the beginning. Moreover, I greatly doubt his assumption that the rupture came suddenly. Now let us examine his rupture, but mind you can see the whole abdomen. Let us put him in a good light and tell him to stand erect and cough. Now you can see that he has a commencing rupture upon the left side, although he is quite unconscious of its existence. If now you tuck up the scrotum with the tip of the finger and explore the left inguinal canal, you will find that the external abdominal ring is large and relaxed and the whole inguinal canal is patulous. When he coughs, a slight impulse can be felt, but not very much. Here, as is almost invariably the case, we prefer the evidence of the eyes to that of the fingers. "The eyes are the windows of the soul." The right inguinal canal and right side of the scrotum are occupied by a swelling which can be seen to become much bigger when he coughs. Now make him lie down. It has gone. The right inguinal canal is very patulous and relaxed, and indeed hardly exists, for the finger can be thrust straight into the abdominal cavity.

In congenital hernia the canal has not been obliterated, and the finger has to travel up it almost as far as the middle of Poupart's ligament before it enters the abdomen. Also the canal is often so narrow that the finger cannot be pushed into it at all.

If now we make this man stand up again, and distract his attention so as to get him to relax the abdominal muscles, we shall see that the hernia is only a part of the fault in his abdominal wall. Outside each rectus abdominis the abdominal wall is bulged into a sort of funnel leading towards the inguinal canal. Could you look within his abdomen, the hypogastric fossæ would have corresponding hollows leading like funnels towards the inguinal canals. Further, the whole of the lower part of the abdomen is bulged, especially over the iliac crests and crural arches. The upper part of the abdomen, on the contrary, is rather flat, or even hollow. If you wished to cure this man of his ruptures it would be necessary not only to repair his abdominal wall, but also to restore his viscera to their proper height. Now in a young man with a congenital or traumatic hernia the only defect in the abdominal wall would be the hole through which the rupture had escaped.

I not infrequently see cases like this man's operated upon. A little while ago I saw a man who had actually been operated upon twice, and had twice relapsed. Such failures are greatly to be regretted, because they bring a good operation into disrepute, and make the public doubt the skill of surgeons. Such errors can easily be avoided by attention to the points which have been exemplified by this case. He will now be ordered a suitable truss. If his hernia was irreducible owing to adherent omentum or other similar causes, it would then be best to operate to enable him to wear a truss, which is very different from doing an operation to cure his rupture.

On another occasion I will show you a case of congenital hernia, and point out more fully why it is so suitable for the operation of radical cure. Be suspicious of patients who are aged and worn, whose work is heavy, who have double ruptures, whose abdominal walls are like those of this man, and whose herniae are obviously acquired and due to pathological causes.

CLINICAL LECTURE

ON

INTRA-THORACIC ANEURISM.

Delivered at Brompton, May 27, 1896, by

C. Y. BISS, M.D., F.R.C.P.,

Physician to the Hospital for Consumption and Diseases of the Chest.

GENTLEMEN.—There is no condition of chest-disease which is more clinically interesting, or deserves more careful study, than that of intra-thoracic aneurism. It is a condition which does not come before us at all so frequently as other chest affections,—indeed one is almost disposed to think that it is increasing in rarity,—but it claims our special attention for two separate reasons: first, its extreme gravity; and secondly, because it may exist in a latent or obscure form, undiscoverable by our art, or only to be made out with difficulty, and yet constitute all the time a most serious danger to life. There is, indeed, no clinical problem which makes greater claims upon our knowledge and practical skill, than that which is presented in the detection and localisation of an obscure aneurism in the chest.

In preparing this lecture, it appeared to me that the subject might be dealt with in various ways. It might be handled systematically, for instance; but such treatment of it would be unsuitable to an audience like the present, and would exceed the limits of our time. It would not be unprofitable, perhaps, if I were to lay before you a selection of histories of aneurisms which had been treated in this hospital, with comments on the cases; nor would you find it uninteresting if I were to show you the remarkable series of pathological preparations our museum contains, as illustrations of the various aspects which aneurisms may assume in their courses and terminations. What I propose to do now, however, is to select a few features of the subject, and those mainly of a practical kind, and to illustrate them as far as I can by brief notes of cases that have actually come under my own observation, and by living cases which I shall submit to your examination at the close of the lecture. I may say that the only form of aneurism

of which I propose to speak is that of the thoracic aorta.

Such aneurisms, whether as fusiform dilatations or sacculations of the aorta, may be (1) Latent, (2) Obscure, or (3) Obvious; and of these three classes the last is, in a sense, the least interesting; for it is, of course, comparatively easy to detect and interpret the physical signs of a large aneurism bulging against the chest-wall, or one giving rise to marked signs or symptoms of pressure upon the viscera, vessels, or nerves in its neighbourhood. Latent aneurisms, also, are of less clinical interest, for the simple reason that they are detected only post-mortem, or when they become obvious. But obscure aneurisms are those which we are in most danger of overlooking when they ought to be found, and which most tax our powers of accurate diagnosis.

Let me give you an example of latent aneurisms. A. S., a woman aged 44, who had borne twelve children, was admitted under my care into the Pollock Ward on the 18th of December, 1895. She had never suffered from rheumatic fever, but her father had died from chronic rheumatism, her mother was a sufferer from cardiac disease, and one brother had heart disease after rheumatic fever. A year ago, when she began to get about after her last confinement, she found she suffered from shortness of breath on exertion, though having never had any previous cardiac symptoms; and this dyspnoea became progressively so much worse, in spite of treatment, that she sought admission to the hospital. On admission the heart was found to be much enlarged, the apex beating behind the sixth rib, two and a half inches outside the normal site. A systolic murmur was audible at the apex, in the axilla, and in the left scapular region. At the aortic base a harsh systolic murmur was detected, alternating with a rumbling diastolic murmur conducted down to the apex; while the second sound was not at all distinct. These were the only noteworthy physical signs in the chest, except that the bases of the lungs behind were rather dull to percussion, and sibilant and bubbling râles were heard there. The diagnosis made was "mitral and aortic insufficiency; considerable cardiac dilatation; loss of compensation following on parturition." The patient improved somewhat under treatment, presenting no new symptoms, and complaining of nothing except a certain amount of

persistent dyspnoea, but on the 2nd of January, seventeen days after admission, she died rather suddenly. At the post-mortem the heart was found much enlarged, its cavities were dilated, the left ventricle being specially dilated and moderately hypertrophied. Both the mitral and aortic orifices were incompetent, but there was no evidence of valvular disease. The aortic orifice opened into an expansion the size of a large orange springing from the posterior part of the arch, forming a large bulging projection, but scarcely a saccule. The whole of the first part of the arch was involved in this bulging. The transverse arch was dilated as far as the junction of the second and third parts. The dilated portion was thickened, irregular, and atheromatous.* I think from this note it will be evident that no other diagnosis was possible than that which was made, and that as all the symptoms and signs characteristic of an aneurism were wanting, it was impossible even to suspect the aneurismal condition actually existing. This may fairly, therefore, be classed as a case of *latent* aneurism.

Among the cases which I have to show you after lecture, there is an admirable example of *obscure* aneurism. The patient, a woman of 50, complains of general feebleness, and pain in the back, localized below the angle of the left scapula, also about the cardiac region, and indefinitely about the upper chest. She has cardiac enlargement and hypertrophy of the left ventricle, with the murmurs characteristic of mitral and aortic regurgitation : there is also a systolic bruit of rough character heard in the aortic area and propagated along the course of the aorta to the left clavicle. The aortic second sound is much accentuated, and almost ringing in character ; and this intensified second sound can be traced along the whole course of the aorta to the mid-dorsal region. There is, however, no definite pulsation in the front of the chest, nor behind, and no dilated veins. The right pupil is somewhat larger than the left, and the right vocal chord is partially paralysed. The occurrence of these symptoms on the *right* side is remarkable. I apprehend that the diagnosis in this case must be that of aneurism, but that the exact localization of the sac is very difficult. It

will be a matter of special interest to you to examine this case after lecture.

I am anxious, in the next place, to make a few remarks about the aetiology of aneurism, and to offer a few illustrations from experience. The cases I have seen at this hospital have included, as in the experience of other physicians, so many cases of aneurism amongst old soldiers and sailors, that I can quite confirm the opinion as to there being causes of a special kind for the occurrence of the disease in connection with these occupations. These causes I take to be complex rather than simple. There can be no doubt that alcoholism, syphilis, and sudden strains are influences which concur in their effects on these patients. The first two act by producing degeneration of the arteries ; the latter by causing the arteries to give way at spots weakened by disease or degeneration. That alcoholism produces arterial degeneration is admitted on all sides ; that syphilis induces endarteritis with degenerative changes is also admitted ; and, if it were doubted, would be readily proved by the significant fact that the occurrence of aneurism amongst women (the sex which suffers least from this affection) is more common amongst prostitutes than other females. In this connection it is interesting to note that the primary lesion may be, as pointed out by Bramwell, in the middle coat, and begin as an obliterative affection of the nutrient vessels in the walls of the aorta leading to a degeneration of the media. As regards strain, I think there can be no doubt that the exertions soldiers have to make in marching and "doubling" with tight uniforms, cross-belts, and knapsacks on, must subject the intra-thoracic circulation to great augmentations of pressure ; and it may be that the improvements that have taken place of late years in the marching "kit," explain the diminishing frequency of aneurism among soldiers : while as regards sailors, the sudden and severe exertions to which they are exposed in climbing, pulling, etc., are too obvious to require notice. One remarkable example of strain has come under my notice, the more remarkable as in this case a sudden and severe exertion was probably the only cause of the aneurism. The patient was a comparatively young woman of about 35, who presented herself in the out-patient department a few years back, complaining of pain in the chest and breathlessness. She had a large aneurism of the

*The above particulars are extracted from the post-mortem records of the Hospital, by kind permission of the Pathologist.

aorta, of which, in spite of all treatment, she died about eighteen months later. The cause of her aneurism was not easily traced. She was hale and hearty; had borne no children, nor had any miscarriages; had led a healthy life, and gave no suspicion whatever of alcoholic habits or of syphilis; but she remarked one day when being closely questioned, that some years before, being very strong and active, she had rather prided herself upon lifting heavy pails in the kitchen and dairy at the farm-house where she worked, and on one occasion even lifted a calf out of its pen in her arms. Furthermore, when her master was very ill, she nursed him with great assiduity and activity, sometimes lifting him, by main force, though a heavy man, from one side of the bed to the other. Upon the whole it seemed to me that her aneurism was probably due to a sudden rent or injury of the vessel produced by sudden and severe strain apart from any other cause. Another very interesting case once came under my notice in the out-patient department which illustrates the cause of a most exceptional case of aneurism, if, indeed, what I am about to speak of was the cause. The patient was a man of about 55 years of age, strong and active, but had an aneurism of the transverse portion of the arch. Below the first rib on the left side was a cicatrix, and in the corresponding supraventricular fossa there was another. In storming a breach in the Chinese war under General Gordon, he leaped down upon a Chinese spear, which pierced the upper part of his left chest but without inflicting a mortal wound. How he escaped more serious injury it is difficult to conceive; and it is an interesting question whether a possible graze of the aorta, or some injury of the tissues in its neighbourhood, had occasioned the aneurism which developed just below the seat of the wound.

ANEURISMAL SYMPTOMS AND PHYSICAL SIGNS.

The symptoms and signs which proclaim the presence of an aneurism are, to put it briefly, those due to the pressure of the aneurism upon surrounding parts; and it may perhaps be useful if I make a few remarks regarding their frequency and intensity in cases which have fallen under my own observation.

SYMPOTMS: 1. *Pain.* This is a very variable symptom, often absent, often of an anomalous character, and probably often felt in situations to

which it is reflected from the point of pressure. The subject of reflected pain is too large a one to discuss at present, but it is important to remember the fact that pain is not always produced in the locality where it is felt. Pain due to aneurism is probably most severe when it is produced by the aneurism pressing a nerve against a vertebra or a rib.

2. *Dyspnoea.* This is a very common symptom indeed; often the only prominent one of a case, and the only one complained of. I can recollect a case in which the effects of aneurismal pressure against a bronchus were mistaken for bronchitis and laryngitis, apparently because the patient had a cough and was hoarse. Perhaps it would not be too much to say that dyspnoea, unless obviously due to other causes, ought always to suggest the possibility of aneurism. It should be remembered also, that dyspnoea of this nature is not necessarily due to direct pressure against the trachea or bronchus; it may be due to pressure upon or stretching of the vagus nerve, or the recurrent laryngeal nerve.

3. *Altered voice.* It is important to remember that, as has been pointed out by Semon, a paralysed vocal chord is not necessarily accompanied by any change in the voice. I can very well remember once noticing a striking example of this in a man who complained of a deep fixed pain in the chest, but had practically no physical signs of disease. It occurred to me to examine his larynx, when I found, that although he spoke clearly and naturally, one vocal chord was paralysed.

4. *Cough and stridor.* Cough is often due to direct pressure upon the trachea, or bronchi, by aneurismal tumours; but in all probability its causation is not always so simple. It may be due to nervous disturbances and nutritional changes, produced indirectly by pressure upon, or stretching of parts. When accompanied by haemoptysis in cases of suspected aneurism, the patient should be kept under the closest observation, and at absolute rest; for slight and repeated bleedings frequently mean that the aneurism is thinning and giving way, and may soon burst into the lungs or air-passages. The *stridor* produced by aneurismal pressure is so remarkable that when once well heard it can hardly be forgotten; but it is frequently absent in tranquil breathing, and only becomes audible when the patient talks, or gets

out of breath, so that the respirations are hurried. When present, it is a most significant sign.

5. *Dysphagia.* This is certainly a rare symptom, and I am disposed to believe that a moderate amount of pressure on the oesophagus may exist without any marked difficulty of swallowing being present. I venture to remind you that when dysphagia occurs in a patient suspected of aneurism, an oesophageal bougie should not be passed, or passed with the greatest caution, for fear of producing rupture of the sac, which is sometimes softened at the point where it presses against the oesophageal wall.

PHYSICAL SIGNS: 1. *Pulsation.* This is a frequent and valuable sign of aneurism, but it may be obscure and even absent. It is best detected by the application of the whole hand to the surface of the chest; or, better still, by the use of the rigid stethoscope; for fine degrees of pulsation can be detected by the impulse thus conveyed, which would escape examination by the hand.

2. *Diastolic shock, sometimes called diastolic jog, and accentuated second sound.* This I believe to be the most valuable single sign of aneurism. I refer, of course, to that peculiar shock which can be felt over the base of the aorta at the moment when the valves close at the end of the ventricular systole. The amount of shock which can sometimes be felt is very remarkable; and the greatly accentuated second sound, which frequently has in these cases a metallic or twanging character, is quite distinct from anything heard in cases of aortic valvular disease, or of high arterial tension, and may fairly be called pathognomonic of aneurism. The cause of this phenomenon is doubtless the increased pressure produced within the aorta by the loss of resiliency of that portion of its walls which is affected by the aneurismal changes.

3. *Bruits,* generally systolic in character, are frequently associated with aneurism, but they are almost as frequently absent, and do not, therefore, constitute a sign of great value, except when they are heard over the back of the chest, as in a remarkable case I have to show you to-day, where the bruit is propagated down the course of the aorta, and about halfway down the spine becomes distributed over quite a wide area of the back,—indicating, as I believe, a considerable degree of aortic dilatation, if no more.

4. *Dilatation of one pupil.*—This is always a remarkable sign when present, but it is frequently absent; and it is well to remember that during the progress of a case it may “come and go,” which would indicate, no doubt, that the internal pressure upon the sympathetic which had produced it, had become relaxed by some change in the position, or pressure, of the aneurism.

5. *Tracheal tugging.*—This sign, which we have been but recently taught to discern, is no doubt one of great value, although by no means of invariable occurrence. It is obtained by placing the fingers on either side of the larynx and exerting gentle traction upwards on the trachea, when a rhythmical series of tugs, corresponding to the beats of the heart, is felt. It would seem to be best made out in those aneurisms which press directly upon the trachea or bronchi; and to be least observable, or not observable at all, in those which involve the first and third parts of the arch, or the descending thoracic aorta. I can demonstrate it to you in one of the cases brought forward to-day.

CASES WHICH SIMULATE INTRA-THORACIC ANEURISM,

It will, I trust, be interesting to you if I briefly describe some cases which have simulated intra-thoracic aneurism under my own observation.

1. An intra-thoracic growth may sometimes strikingly resemble aneurism, and indeed be indistinguishable from it. Six years back I was consulted by a gentleman, aged 57, an accountant by profession, who complained of cough of six months' duration, a sense of weight at the chest, and dyspnoea, which became severe on even slight exertion. There was slight expectoration in the morning, and occasional slight haemoptysis. His case had been diagnosed by a physician some little time previously, as one of asthma. On examining him the first thing I was struck with was the stridor that accompanied his inspirations, which though of slight degree was quite characteristic of pressure somewhere upon the air-passages. His chest was rather emphysematous. At the right apex there was found some dulness, and the breath sounds were faint and of bronchial character. Over the corresponding base the breathing was almost inaudible. At the aortic cartilage the second sound was peculiarly accentuated, and a fine to-and-fro bruit was heard. Slight pulsa-

tion was noticed in the second and third spaces to the right of the sternum. Above the right clavicle there was pulsation. The diagnosis of aneurism of the first part of the arch was made, and perfect rest enjoined on the patient. Some weeks later I saw him in consultation with his medical attendant, and found the signs much the same, except that the whole of the right base had become dull. A tentative aspiration with a small syringe drew off serous fluid. The next day he was tapped by a surgeon, but no fluid could be found—a result which it is difficult to explain in view of the facts just mentioned. Without the development of any fresh signs the patient gradually declined in health, and died some weeks later. At the post-mortem, which was made by his medical attendant, who kindly furnished me with a note of it, a malignant growth, which proved to be a lympho-sarcoma, was found pressing upon and almost encircling the right bronchus. There were atheromatous changes of, and about, the aortic valves. The right lung was partially collapsed. The physical signs were thus completely explained; but it seems hardly possible to have avoided the diagnosis of aneurism, although, as the sequel shows, this proved to be fallacious.

A case of fibroid disease of the upper lobe of the right lung accompanied by contracting changes, simulating an aneurism of the first part of the arch. I can remember a case which occurred a good many years ago, though I have not at present an exact note of the particulars, which offers an interesting illustration of how aneurism may be simulated by certain other conditions. The patient was a man of middle age, and I think had formerly been a soldier, who complained of pain in the chest, slight cough, and some shortness of breath. He did not look ill, and was not wasted. On examination, the right apex was found deficient in expansion and dull to percussion, especially towards the inner margin of the lung, where also there was detected obscure heaving pulsation. A systolic bruit accompanied the first sound at the aortic valves, and the second sound was much accentuated in the same locality. At the apex of the right lung the breath sounds were absent. There were no other important symptoms. The diagnosis of an aneurism of the first part of the arch of the aorta was made, and the patient placed on a course of treatment by rest and iodide

of potassium. No change took place in his condition until about a fortnight later, when he suddenly developed convulsive movements of the right hand and arm: the next day he became dull and stupid, and the comatose condition progressively deepened till he died. At the post-mortem examination an abscess was found in the left frontal lobe. The upper lobe of the right lung was the seat of extensive fibrotic changes, and contained several bronchiectatic dilatations, from one of which the septic matter had doubtless been derived which was the cause of the cerebral abscess, as is often the case in chronic pneumonia associated with bronchiectasis. The right upper lobe being much contracted, the first part of the aortic arch had been uncovered; and this explained the pulsation that had been felt in that neighbourhood, and, to some extent, the intensification of the second sound. The aorta itself was atheromatous in the neighbourhood of the valves. The singular point in this case was the quiescence of the lung-signs and symptoms. The cough was slight, and scarcely complained of, and there was little or no sputum; while the dulness at the inner side of the right apex being accompanied by obscure pulsation, seemed not unreasonably attributable to an aneurismal dilatation of the aorta, or the base of the innominate artery, especially as there was an aortic systolic bruit present, and a much intensified second sound.

I will briefly quote a third case, not very dissimilar in character. A man of middle age came complaining that shortly before, after climbing a hill, he had brought up a few mouthfuls of blood. He had no cough, no hereditary tendency to tubercular disease, but a clear history of syphilis a few years previously. On physical examination a little dulness was found at the inner side of the apex of the right lung, where distinct heaving pulsation could be detected, with intensification of the second sound. I do not remember that there were any other physical signs, but I have not now a note of the case to refer to. Some months later I saw the patient again, and now the nature of the disease was obvious, for he had all the signs of a small cavity in the upper lobe of his right lung. The signs originally detected must have been due to some indurative consolidation of the inner part of the apex accompanied by sufficient contraction to partly uncover the root of the

aorta, which would be quite sufficient to explain the incidence of the pulsation felt and the intensified second sound heard in that situation.

Before leaving this part of the subject, I would like to make a reflection that occurs to me in the review of these cases. You will remember that when speaking of physical signs I remarked that there was a peculiar intensification of the second sound at the aortic base, generally accompanied by a diastolic shock, which could be felt as well as heard; and that this is probably the most important, as well as the most constant, SINGLE sign of aneurism. Now in none of these three cases was this sign present: the second sound was intensified in all, but it did not possess that peculiar character that is characteristic of aneurism—a character the discernment of which can only be acquired by clinical experience. I cannot help thinking, however, that if more attention had been paid to this point, it might have been suspected, if no more, that these cases, though strongly suggesting the diagnosis of aneurism, were not really examples of that disease.

Treatment.—I will conclude my remarks with a few brief observations about treatment, although I have no new suggestions to offer. I believe that only those aneurisms of the aorta which have narrow openings into the sac admit of any hope of cure by the solidification of laminated layers of coagulated fibrin: for in these cases alone is it possible to promote that stagnation of the circulation which favours the deposit of fibrin. The only treatment that affords any reasonable hope of success in this direction is the Tufnell treatment, the essentials of which are absolute rest in bed and the reduction of the diet, both in solids and liquids, to very limited proportions. The radical principles of this treatment appear to me to be sound. The rest and modified starvation reduce the tension of the circulation, and render the deposition of fibrin more easy. I think that in every case, the risks of his condition, and the nature of the plan of treatment, should be fully explained to the patient, so that his full and intelligent concurrence may be obtained; otherwise the hope of success is very small. I always give iodide of potassium, and entirely agree with those who believe that this remedy relieves aneurismal pain, and increases the coagulability of the blood. I have never seen an aneurism cured, nor bene-

fited, by the introduction of wire, &c., into the sac; and I am disposed to regard this form of treatment as not only useless but dangerous, especially by exposing the patient to the special risks of embolism. Lastly, I believe it is possible sometimes to benefit an aneurism by venesection; and in cases in which the tension of the circulation is high, the value of this form of treatment ought to be borne in mind. In the later stages of aneurism it is possible sometimes to prolong life by reducing the pressure which the aneurism is exerting upon the air-passages and nerves by the withdrawal of blood from the arm. About two years ago I was called one night to a patient who was almost moribund from the pressure of an aneurism of the transverse arch upon the trachea and left bronchus. As the case seemed desperate, I advised venesection; a surgeon was summoned, and thirteen ounces of blood were withdrawn from the arm, with wonderful and immediate relief to all the symptoms. The patient's life was prolonged, with comparative comfort, for twenty-four hours longer. The remedy, however, is one that we can only resort to in unusual cases, and extreme emergencies.

A NOTE FROM THE CLINIC

OF

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Locomotor Ataxia.

THESE are three cases of locomotorataxia which have all been attending as out-patients for some months. One of them, in a man æt. 52, is of fifteen years duration, and has been in a stationary condition for some years. The other two, both in men of about 43 years of age, are early and as yet slight cases.

In all three of them there is a history of antecedent syphilis. This occurs in such a high percentage of cases of locomotor ataxia, and so much more frequently than in other forms of degeneration of the spinal cord, that there is apparently a definite relationship between the two diseases. Much the same connection, though the

influence of other factors, such as work, strain, and high tension, is probably necessary, holds between syphilis and general paralysis of the insane. In both cases the effects (ataxia and general paralysis) are generally a very late result of the primary infection—so much so, indeed, that these two diseases have been regarded as being the manifestations of a fourth ("post-tertiary") stage of the disease. These "quarternary," like the tertiary manifestations, may never occur, and very probably some inherent or acquired weakness of the nervous system is responsible for the development of locomotor ataxia in a syphilitic subject. It is interesting to note as showing an association between the two diseases that cases of general paralysis of the insane may after a time become arrested, and remain stationary with many of the symptoms not of the original disease, but of locomotor ataxia.

The pathology of locomotor ataxia may be summed up by saying that it is a degeneration of the sensory nerve fibres. This degeneration, which may be due to a poison or toxin generated as the result of a syphilitic process, probably attacks the trophic ganglion cells in the posterior root ganglion. These nerve cells have a T-shaped process, the branches of which run centrally into the spinal cord, and peripherally into the sensory nerves. Degeneration commencing in these cells will therefore spread in the two directions indicated, leading to peripheral neuritis on the one hand, and to an ascending degeneration in the spinal cord on the other. This ascending degeneration is first seen in the postero-external column, and subsequently, viz. higher up, in the postero-median or Goll's column. As a result of this ascending degeneration, the nerve fibres atrophy and disappear, the spinal cord therefore becomes smaller or wasted, and from this feature the disease is often spoken of as *tabes dorsalis*. The degeneration and disappearance of the active functional nerve-fibres throws into prominence the supporting neuroglia. This apparent increase in the amount of the supporting connective tissue of the tracts affected may be described as a process of fibrous substitution. Possibly the supporting tissue does to some extent increase in amount from the fact that it now has an undiverted blood-supply. This fibrous substitution is generally spoken of as sclerosis; but it must be borne in mind that it is rather passive than active, that it is not the

result of inflammation, and thus differs from the increase of fibrous tissue, or fibrosis seen in cases of transverse myelitis, where there is a chronic diffuse myelitis. The pathological changes are then an ascending or systematic degeneration in the cord—systematic because it affects a definite tract of nerve-fibres having the same function—and a peripheral degeneration of the nerve-fibres. To call this, as is often done, a neuritis is inaccurate, for there is no primary inflammation. The termination "*-itis*" has acquired the significance of inflammation: originally, however, it had no such meaning, being only an adjectival termination agreeing with *nosos* (disease) understood, so that, strictly speaking, neuritis would mean disease of the nerves and not necessarily inflammatory disease; but gradually in most organs the disease par excellence being inflammation, it has become synonymous with inflammation.

The symptoms are those due to changes in the sensory nerve apparatus. The reflex arc is broken and the knee-jerks, as in peripheral neuritis, are lost. Primary atrophy of the optic nerve occurs in locomotor ataxia, and with this it is well to associate the Argyll-Robertson phenomenon, or loss of the pupil reflex to light, while the pupil continues to react in accommodation. Paresis, or paralysis of the ocular nerves, leading to ptosis or to squint and diplopia, may occur, but with this exception the motor nerves are remarkably free from degenerative processes. Impairment of tactile sensation is common; the patient feels the ground imperfectly and with difficulty; as a result it may seem to him that he is standing not on *terra firma* but on something woolly, or on a cushion. In like manner sensation of pain is modified, being delayed, imperfectly localized, wrongly referred, or absent.

Sharp darting pain in the legs—lightning pains—are a frequent and often the chief symptom that brings the patient to the hospital. Pains of varying character in the limbs, back, and abdomen may resemble those of rheumatism, sciatica, or lumbago. Thus what at first appears to be rheumatism may eventually turn out to be the pains of *tabes*. Double sciatica should at once arouse a suspicion of some central change in the cord, or of pressure on the cauda equina.

The loss of muscular sense, the result of degenerative changes in special nerves of muscular

sense, is a most important and characteristic feature of the disease, and is responsible for the loss of muscular co-ordination, or ataxia. It accounts for the characteristic staggering gait, for the inability to stand with the eyes shut, or to walk in the dark. You notice that when told to shut his eyes the patient begins to shake and sway, and if a more severe test is then applied, viz. standing on tiptoes, that he falls—usually backwards. Patients often tell you when questioned that when they wash their face, and for this purpose close their eyes, that they fall forwards, or take an unpremeditated "header" into the basin. As long as they can watch their own movements, and thus keep themselves informed of the proper direction and amount of muscular contraction to be maintained, they are fairly safe. But if a man with ataxia goes out at night his eyes can no longer vicariously perform the duties of the nerves of muscular sense, and his staggering gait may land him in the cell intended for the drunkard. Locomotor ataxia is rare in women, but if an ataxic nurse while holding a baby does not keep an eye on her arms, her burden will be in danger of being dropped. It is rare, but sometimes in peripheral neuritis, and it is said more commonly in diabetic neuritis, the muscular sense is similarly affected. This condition has been called "pseudo-tabes," and is in fact ataxia of the lower extremities, the eye and the visceral symptoms being absent; it is in this way distinguished from the more complete disease.

Trophic lesions, such as perforating ulcers of the feet, or Charcot's disease of joints (arthropathy) seem to point to degeneration in the nerve fibres or centres directly concerned. Atrophy of bone produced in the same way is a cause of spontaneous fracture.

Visceral crises or sharp attacks of pain comparable to the lightning pains in the legs form an integral part of the disease. Gastric crises, or attacks of sickness and pain, intestinal crises—colic and diarrhoea,—rectal crises or tenesmus, renal crises simulating renal colic, and other forms, may occur. These crises may imitate organic disease; thus a case of gastric crisis associated with the constipation which is so common in tabes may, if the nature of the primary disease be unknown, be thought to be one of intestinal obstruction. These crises are presumably due to changes in the sym-

pathetic of the same nature as those in the spinal nerves.

Constipation is generally present. Bladder symptoms—retention or incontinence—are usually present, and are of considerable importance, since cystitis, pyelitis, and suppurative nephritis may thus be set up and prove fatal.

The treatment of tabes is unsatisfactory, inasmuch as it is chiefly symptomatic. There is no drug which has a definite effect for good on the primary lesions. Mercury and iodide of potassium have been discovered as specifics for the secondary and tertiary manifestations of syphilis, and though the latter remedy is generally given, and I have given it to these three cases of tabes, it cannot be said that it does any real good. We have yet to see whether any specific for tabes and general paralysis, which have been spoken of as the manifestations of a fourth stage of syphilis, exists. Gold, silver, and suspension have been tried in the balance and found wanting. Rest in bed, and the constant performance of planned exercises with the feet, are employed apparently with success by Erb, but the treatment has not yet been generally adopted. Pain seems to be fairly relieved by antipyrin or phenacetin, and constipation by ordinary remedies. Cystitis I have found to yield very satisfactorily to salol given in a quinine mixture; after being taken the salol is broken up into carbolic and salicylic acids, and is excreted by the kidneys. It thus washes out and disinfects the urinary passages from above. This, together with careful catheterisation, has kept the urine normal in a patient with spastic paraplegia, who more than two years ago came here with ammoniacal urine.

Hemiplegia after Sunstroke.

This young man, æt. 22, is suffering with rigidity and some loss of power in the left arm and leg, exactly like that which results after cerebral haemorrhage. The reflexes are exaggerated, and there is well-marked ankle clonus, due to interference with the normal inhibitory action of the encephalon. After being exposed to the hot sun on Derby Day last year he had a fit, and when brought to the hospital shortly afterwards he was found to be suffering from hemiplegia. There was no morbus cordis, no renal disease or syphilis, and it was regarded as being the result of sunstroke. When recovered from, sunstroke leaves its mark on the

central nervous system in an excitable condition ; there is great intolerance to heat, and general instability leading to loss of mental power and balance, epilepsy, and even insanity, but is not usually recognised as giving rise to hemiplegia. There was, however, a somewhat similar case in the hospital last year, and I have heard of another. If the hemiplegia be, as it appears, due to sunstroke, the most plausible explanation seems to be meningitis and inflammation of the underlying cerebral cortex in the motor area, which by destroying the motor cells has led to a descending degeneration or sclerosis of the corresponding pyramidal tract ; the explanation of the "sclerosis" being the same as in tabes, namely a primary degeneration of the nerve fibres or axis-cylinder processes of the degenerate ganglion cells, and a resulting fibrous substitution on the part of the neuroglia.

Here, then, is a systemic degeneration involving the descending or motor (pyramidal) tract, interfering with the transmission of impulses from the cortex, and so giving rise to paresis. The inhibitory action of the higher centres is cut off, as shown by the increased deep reflexes and ankle clonus. There is no interference with the sensory tract or with sensation. The nutrition of the muscles, except for some wasting from disuse, is maintained. This shows that the trophic ganglion cells in the anterior cornu of the spinal cord are intact. If, as happens in amyotrophic lateral sclerosis, they were affected as well as the lateral column, there would be in addition marked muscular wasting.

These two cases, both of systemic degeneration, one in the sensory, the other in the motor tract, form an instructive contrast.

REVIEWS.

The Phonographic Record of Clinical Teaching and Medical Science. Issued by the Society of Medical Phonographers.

The May number of this journal includes a very practical paper by Mr. Peyton Beale on the art of passing catheters. He emphasises the necessity of knowing where the point of the instrument is pressing, says that force is never to be used, and

urges the tyro to neglect no opportunity of passing a metal instrument on the dead subject. Gum-elastic instruments are condemned, and those of soft rubber or celluloid commended. After being cleaned with soap and water and rinsed through with a 1—20 solution of carbolic acid, they are to be kept in a saturated boracic solution till required. In using a male catheter the patient should lie quite flat, and the left lateral position is recommended in the case of females.

Dr. Atchley, of Bristol, contributes a remarkable case. A strumous man, 27 years old, beginning with typhoid at the age of 7, successively developed angular curvature of the spine, phthisis of both lungs, disintegration of the right hip-joint, and left lumbar abscess, in addition to abscesses in the groin, right hip, and right loin. Pieces of bone, apparently from the vertebrae, were spat up, and fluid injected into the right hip is tasted in the mouth in a few minutes. There was no family history of phthisis, and the case is most encouraging as showing what assiduous care may do to prolong life and relieve suffering under trying conditions. Dr. Leonard Rogers suggests that the paralysis which sometimes attacks divers on coming to the surface is due to intravascular blood-clot in the vessels of the spinal cord or brain, brought about by the liberation of excess of the carbonic acid gas in the blood ; and on this hypothesis he proposes the inhalation of oxygen gas as the best remedy for the symptoms.

Researches into the Anatomy and Pathology of the Eye. By E. TREACHER COLLINS, F.R.C.S. (Lewis). Price 6s.

This book essentially consists of the lectures delivered before the Royal College of Surgeons in 1894. Mr. Collins has established such a position for himself as a specialist on all that pertains to the eye, and withal such a name for accuracy of observation and description, that his work is accepted now almost without question. The work before us is destined to form for some time the high-water mark of knowledge on the arcana of eye-specialism. Chapter I deals with the development of the suspensory ligament of the lens and of its capsule ; Chapter II with the histology of zonular cataract ; the development of the vitreous, &c. ; Chapter III with the iris chiefly and origin of the aqueous ; Chapters V and VI with the theory of glaucoma ; and Chapter VII with various traumatic eye troubles. To the general practitioner, and even to hospital teachers, the book must be, we cannot say useless, but beyond all his requirements ; but to the specialist it is absolutely essential, for the information in it cannot be obtained anywhere else.

THE CLINICAL JOURNAL.

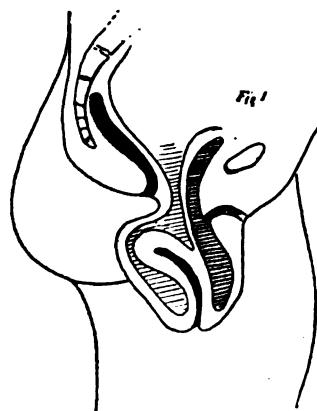
WEDNESDAY, JULY 1, 1896.

A CLINICAL LECTURE ON PROCIDENTIA UTERI.

Delivered at Charing Cross Hospital, May 26, 1896,
By AMAND ROUTH, M.D., B.S., M.R.C.P.
Obstetric Physician with care of Out-Patients, and
Lecturer on Practical Obstetrics and Gynaecology
to the Hospital.

GENTLEMEN,—I have chosen for to-day the subject of *procidentia uteri*, which is one of the commonest displacements of the organ, and the consideration of it, as opposed to *prolapsus*, will be quite sufficient for one lecture.

First I show you a diagram illustrating a very early variety of procidentia, where the cervix is just beginning to protrude through the vulva, and, as we shall see later, it is extremely important to be able to distinguish what the exact condition then is. Supposing the procidentia to be complete, one notices certain points about it (see fig. 1).



First of all, it gets expelled or protrudes from the vulvar orifice, just as far as the vagina wall will allow it. In a typical case of retroversion with procidentia, the uterus is felt to be entirely inside

VOL. VIII. NO. 10.

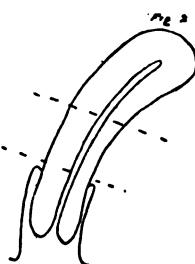
the prolapsed mass. When the uterus is completely outside the vaginal orifice, the peritoneum comes down in front and behind, forming a large pouch in both places which may contain folds of bowel. The posterior cul-de-sac generally contains the ovaries and Fallopian tubes also. It is extremely important to remember that the peritoneum is there and that the bowel may get there also, because one may get cases where the bowel is adherent, and where in pushing back the bowel you may injure it. It needs handling very gently. The mucous membrane of the inverted vagina gets dry and comes to resemble skin, and may become denuded of epithelium, a chronic ulcer sometimes forming, not on the sides where the thighs would rub against it, but generally in front of the cervix, just where the cervical and vaginal mucous membranes join. It may be due to the secretion from the cervix, or may accompany ectropion, or it may be due to urine trickling down the anterior vaginal wall. The urethra, which, in the ordinary way goes straight up underneath the pubic arch, becomes U-shaped and doubled over, so that a catheter would pass up half the distance and would then have to be curved down to get into the bladder. The inverted vagina, which comes down in these cases, draws down the bladder in front, and is therefore always called a cystocele. Prolapse of the posterior vaginal wall does not necessarily contain the rectum, as the rectum is much less closely bound to the vagina than in the case of the bladder, and the result is that the posterior vaginal wall may come down and form a considerable tumour below the vulva, yet with the rectum perfectly unaffected. One other point in the anatomy is, that if you think of the connection of the ureters to the cervix and to the broad ligaments, you will see that the ureters must come down, and be dragged down in order to reach the base of the bladder. So that in any case of procidentia the ureters are very much stretched, and that is why, in some of these cases, there are urinary troubles over and above those due to cystocele.

Supposing the cervix protrudes from the vulva,

as shown in this diagram, it may be due to one of six causes :

First, it may be due to a prolapse of the uterus *en masse* (Fig. 1), with the vagina inverted more or less, especially anteriorly, and the cervix protruding a considerable distance, and we may find the uterus completely outside the vulvar orifice, in a position of retroversion or retroflexion. Now and again the uterus may be in a state of anteversion or anteflexion, and yet completely outside the vulva, but that is very rare. One of the commonest varieties is that which I have described, where the uterus is retroverted or retroflexed and the whole organ is bodily prolapsed down, that is to say, the fundus uteri has prolapsed as much as the cervix, and was probably the first part of the uterus to fall.

It sometimes happens, and much more commonly than books state, that although the fundus has remained practically on its proper level, the cervix is much lower than it should be. This constitutes our second group, and is due to a stretching of the supra-vaginal cervix (Fig. 2).

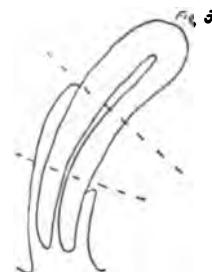


You can understand that in a case such as is here illustrated, that if the supra-vaginal cervix stretches and the fundus remains where it is, the result would be the pushing down of the vaginal cervix, and with it the two cul-de-sacs. So that the cervix may even be driven down and protrude from the vulva.

The third group is similar to the second, except that the elongation of the supra-vaginal cervix is due to true hypertrophy and not to mere stretching. Here the physical effect on the cul-de-sacs are as in Group 2, although the vaginal walls do not first prolapse, as happens if the elongation of the supra-vaginal cervix is due to stretching.

The fourth and fifth groups are due to stretching or hypertrophy of the *pars media* (Fig. 3). Here

the part hypertrophied is that which lies between a line drawn across the uterus from the level of the anterior cul-de-sac and one drawn from the posterior. The result of this state is that the



anterior cul-de-sac is depressed towards the vulva, whilst the posterior cul-de-sac remains at its normal level, or may, in cases of true hypertrophy, be even elevated.

The sixth group comprises elongation of the infra-vaginal portion of the cervix, which is, so far as I know, never due to stretching, but always to true hypertrophy (Fig. 4). It may protrude right through the vulva, sticking out like a finger. It must be remembered that two or more of these conditions may be present at the same time.

Causes of Procidentia uteri.—(First group). Procidentia is predisposed to by parturition, and by laborious occupations, indeed, anything which causes straining. Given one or other of those conditions, there are certain causes which almost inevitably lead to prolapsus. The first is a faulty pelvic floor, the anatomy of which we have recently considered. Another cause is laxity of the uterine ligaments. The round ligament plays little part in keeping the fundus forwards, the chief ligaments which support the uterus are the broad ligaments and the utero-sacral ligaments. You know that when a healthy woman is in the erect position, the uterus lies nearly horizontal, and it is kept so by the cervix being retained upwards and backwards by the utero-sacral folds, which run almost vertically from the uterus, just above the posterior vaginal cul-de-sac to the second piece of the sacrum. If anything relaxes those ligaments, the first effect is that the cervix falls forwards and downwards, rendering it very easy for the uterus to become retroverted.

Retroversion, therefore, is a strong predisposing

cause of procidentia. Supposing the uterus is normally horizontal, the vagina is more or less in a curve which follows pretty closely the pelvic curve. So long as the uterus is anteverted, any intra-abdominal pressure comes to be mainly on the top and posterior aspect of the uterus, and tends to drive it more against the bladder and other anterior structures, which afford a considerable obstacle to prolapse. But the moment it loses its anteverted position and gets more and more erect and into the vaginal axis, then the intra-abdominal pressure takes effect in the direction of least resistance, viz. along the vaginal tube.

That leads me to speak of intra-abdominal pressure as a cause of prolapse. If the intra-abdominal pressure is increased, the effect on the uterus, if the utero-sacral ligaments are faulty, is well marked, for the moment the uterus becomes retroverted the pressure from above is not only more in the direction of the pelvic axis, but is applied to the anterior surface of the uterus, and there is nothing in the new position of the fundus to prevent it being gradually depressed into Douglas's pouch. Therefore, retroversion plus increased abdominal pressure is a common combined cause of progressing prolapse.

I ought next to mention increased weight of the uterus. It often happens that the uterus is subinvolved, and usually the ligaments partake of this; so that if the uterus is too heavy from the sub-involution and its ligaments are too lax to support it, and especially if there is a little yielding of the pelvic floor, you can see at once how readily the womb will come down.

Another cause is ruptured perinæum and injured vagina. Some place too much stress on ruptured perinæum, and others too little. The perinæum must be looked on as the keystone, the triangular body of the posterior vaginal wall. If this perinæum were torn through, first of all there would be less support from above, secondly, the anterior vaginal wall is not able to rest on the posterior vaginal wall, and the tendency is for a cystocele to occur which soon drags down the uterus. You will also remember that there is a sphincter of the vagina and a sphincter of the anus. The external sphincter ani arises from the tip of the coccyx, encircles the anus, and is inserted into the central point of the perinæum. The

sphincter of the vulva is also attached posteriorly to the central point of the perinæum. When the perinæum is torn through, the sphincter vaginæ is torn through also, and the central part of the perinæum thus loses one of its anterior attachments, and the sphincter ani is able to draw back the central point of the perinæum, which makes the vaginal outlet gape, and cystocele becomes thus even more likely to occur.

Next as to *symptoms*. One need not say much about acute procidentia, for it is rather rare. It is due to any such increase in abdominal pressure as may occur in epilepsy, or as may accompany sudden falls, where the uterus is bodily forced through the vagina, and the symptoms, as contrasted with those of ordinary procidentia, are comparable with those of acute as against chronic hernia. The pain of acute procidentia is violent, and of sudden onset; the patient vomits, and generally faints. There may or may not be haemorrhage, but there is usually retention of urine. Those symptoms are sufficient to show that it is acute. But in the chronic cases of procidentia it is wonderful how little the discomfort is. A patient may have a procident retroverted uterus, but exhibit very few symptoms. Many of you have seen women in the out-patient department with the uterus completely outside the body, and yet they only complain of the mechanical trouble; they have but little of the backache and leucorrhœa which occur in slighter cases. In the usual run of cases it is the beginnings of the prolapse which are the most painful. After a time the ligaments and pelvic floor become tolerant to the altered position of the uterus, and the patient's discomfort is lessened. The first symptoms are usually backache and bearing-down pains, with dragging in the loins and groins, generally associated with leucorrhœa. There may be, also, and generally is, irritability of the bladder, due partly to cystocele, and partly to the dragging of the bladder backwards by the retroversion. Leucorrhœa is a prominent and early symptom, and the function of the uterus is generally impaired; so that dysmenorrhœa and menorrhagia may follow.

These patients generally complain of reflex dyspepsia, or diarrhoea, or headache, or depression of spirits; something superadded to the ordinary local symptoms.

I will next say a few words about the *physical signs*. At first one would see the cervix just beginning to protrude from the vulva. After a time a large mass comes out, and one sees at once that the mucous membrane is altered in character and has become rather dry. It has to be distinguished first from a fibroid polypus of the uterus, and secondly from an inverted uterus. The way we distinguish them is by the presence of the external os at the apex, with some ectropion from dragging on the vaginal walls. A fibroid polypus is generally narrower nearest its origin, and can be traced upwards to the endocervix. Its mucous covering is moister and bleeds readily. This, of course, is in strong contrast to the dry mucous membrane of procidentia. Then an inverted uterus is certainly much more spongy-looking and moist than either polypus or procidentia, and it will at once occur to you that there is no orifice of the external os to be seen anywhere, and that instead the openings of the Fallopian tubes may sometimes be seen. But these distinctions are obvious, and the main point which has to be decided is to which of the six varieties of prolapse is the protrusion of the cervix due? If the case be one of the first group (Fig. 1) the retroverted fundus is felt to be inside the procidented mass, and the sound confirms that view.

Supposing it is one of the elongations of the supra-vaginal cervix (Fig. 2), the increase is above the two cul-de-sacs, and would force these cul-de-sacs and the vaginal cervix downwards, so that gradually the vagina becomes shortened, as ascertained by the finger. Taking care to exclude pregnancy, which is not impossible in these states, the sound may be passed, and perhaps enters five inches.

Then one has to distinguish between the elongation of the supra-vaginal cervix being due to stretching or to true hypertrophy. Supposing the sound goes in five inches, the next thing is to replace the uterus—to push it back into its proper place. If it is simply a stretching, you will find that the uterus will at once only measure $3\frac{1}{2}$ inches, or perhaps even goes back to nearly normal in a few minutes; whereas if it is true hypertrophy the elongation will persist for days, or even weeks, and will only gradually lessen under appropriate treatment. Much the same applies to the hypertrophy

and the stretching of the pars media (Fig. 3), but the swelling being above the anterior and below the posterior cul-de-sac, the result is the forcing down of the anterior cul-de-sac, while the posterior remains the same. So that when the finger is passed into the vagina, the anterior cul-de-sac is found to be lowered almost to the vaginal outlet, whilst the posterior cul-de-sac is quite as high as one can reach; quite different, therefore, from the other two cases.

The diagnosis of the last variety—elongation of the infra-vaginal cervix (Fig. 4)—is perfectly



easy, especially when the cervix protrudes from the vulva. The examining finger encircles the cervix, and the cul-de-sacs are at their normal level.

Next as to *treatment*, in connection with which I will first mention prophylaxis. In the prevention of procidentia, one must look after the proper management of childbirth. Care should be taken not to use any undue force; to prevent, by counter-pressure and drugs, a too rapid delivery, and so try to avoid laceration. If a tear occurs, it should be restored at once, or if from the nature of the case this is impossible, the patient should be put under such circumstances as will cause the least possible tension on the part. For instance, sometimes one is able to make out that some muscular fibres of the posterior vaginal wall are torn above the perineal body. If so you can keep the patient on her back a little longer than usual, and take care that her rectum is not allowed to get full, for if the patient becomes constipated during the first week of her lying-in period, the rectum gets loaded, the divided muscular fibres are forced apart, and a rectocele almost certainly results. Also after much stretching of the anterior vaginal wall you must take care that the catheter is passed at regular intervals to prevent any bulging down, and see that the uterus is involuting properly and the patient kept in bed longer than

usual. The uterus must also be kept at a good level. I try to examine patients at the end of two or three weeks, when they get up, and if the uterus then shows a tendency to retrovert, it is well to put in a Hodge, or keep the patient at rest till involution is complete.

If a patient has procidentia, what are we to do? There are constitutional remedies, such as tonics, and the treatment of any diathesis, and care must be taken to give purgatives, for straining increases the intra-abdominal pressure, which must be avoided until the ligaments and muscles recover their tone.

Regarding local treatment, a procidented uterus must of course be reduced before treatment can be begun. It is not simply that you put in first the part which came out last, for this may be done and yet the patient will be in much pain afterwards. What one has to find out is: what is the condition and position of the uterus itself? If it is in a state of retroversion, and you simply reduce it *en bloc*, you leave the uterus retroverted and in a position where it is not so comfortable as formerly. Therefore you must take care to put the uterus in an anteverted position. It also very often happens that one or both ovaries are prolapsed down into Douglas's pouch too, and if you put the uterus back without anteverting it, these ovaries are almost sure to be bruised. So that it is most important to pass the sound if you cannot otherwise antevert the uterus.

If the fundus has all along remained at its proper level, all you have to do is to push the rest of the uterus up, and keep it in by a suitable support. Having reduced it, the next step is to keep the organ in position. This may be done by medicinal or mechanical pessaries, or by some form of operation. Pessaries of tannin or alum and cacao butter are mere temporary expedients for constricting the vagina after reduction. Most pessaries merely prevent the uterus from coming outside; they do not keep it in its proper position. A ring put into the vagina distends and shortens it, and keeps the uterus really below its proper level. Still, so long as a ring will keep in, it is about the best thing we can use for these cases. But it often happens that the outlet of the vagina is larger than the upper end. If one examines the case properly, it is frequently found that although there is a wide outlet the ring will find a resting-

place at the top of the vagina, that there is a constriction halfway down which enables the ring to stop in; and one may often succeed with a small ring where one has failed with a large one. There are many other varieties of pessaries which suffice to keep the uterus from coming outside. Air-ball pessaries are sometimes useful, where one cannot get anything else to keep in. They are put in closed and are then inflated with air, or air and water, for there is a tendency for the air to escape at the joint, and escape is avoided if a little water be put in also. They take their support from above the sacro-iliac ligaments, and they answer very well if there is not much stretching of the anterior vaginal walls; so also does this Priestley's or Napier's cup-and-stem pessary, but I think the best is Napier's, as it is flexible.

I show you also some varieties of Cutter's pessaries for use when the vaginal walls are very relaxed. They are fastened to an abdominal band. Zwanke's pessaries also are useful, but are unsuitable if a cystocele or rectocele is present. I have known cases where this mushroom-shaped instrument will keep in when nothing else would, as the vaginal walls seem to grip the top of it. Again, Sinclair Coghill's is a useful form in some cases where there is some support from below, yet there is no perineal body. This anteversion pessary of Galabin is useful where there is cystocele. The uterus is reduced first, the pessary is then put in, and the cystocele rests on the anterior brow. None of these will keep the uterus at its proper level, except the last perhaps, but they will prevent it protruding. The best pessary for keeping the uterus at its proper level is a Hodge; there is nothing to equal it. The Hodge may have bars across it anteriorly (Greenhalgh's) to prevent the descent of the cystocele; it is of very little use where the cervix is atrophied, and also if the perineum is deficient, because the moment a patient strains or moves about, the posterior bar gets in front of the cervix and it is soon expelled. Supposing the structures in Douglas's pouch are tender, the upper end of the Hodge should be invested with a glycerine pad.

If a Hodge fail, some form of figure-of-8 pessary is useful—shaped from an ordinary round malleable ring. These rings are made in different sizes, and the length of the vagina having been ascertained, the figure-of-8 pessary can be easily moulded. If

there is a marked cystocele, it is better to have the anterior ring tilted up, so that the cervix can lie in one ring and the cystocele on the other. A shape which is useful is a sledge shape (Fig. 5); in many cases this will answer best.



Next as regards operations. A large number of operations are done for this purpose. First of all, operations on the vulvar outlet. If the perinæum be deficient, it is necessary to restore its integrity by performing an ordinary perinæorrhaphy. It is extremely advisable to do that before the prolapse is very well marked. If you find the perinæum is badly torn, that the uterus is coming down, and that an ordinary support cannot be kept in, it is far better to restore the perinæum and so enable the patient to wear a small ring or a small Hodge; or the vagina may be narrowed, either anteriorly or posteriorly. The anterior operation is required in cystocele, the posterior in cases of rectocele, or prolapse of posterior vaginal wall; but the operation which is most suited to every form of procidentia is a combined posterior colporrhaphy and perinæorrhaphy, turning up a big flap along the posterior vaginal wall, and so narrowing both the vaginal outlet and the vagina itself. In this operation the flap serves to strengthen the new perinæal body, nothing having been cut away. Even in performing anterior colporrhaphy for cystocele nothing should be cut away, but the stripped-off mucous membrane should be everted, and drawn up into a ridge by fixing it to a bone or celluloid scaffold. It may be necessary to curette the uterine mucosa if the uterus is bulky and heavy and catarrhal, and if the vaginal cervix is elongated it is advisable to amputate the cervix by Schroeder's method. Sometimes I amputate the cervix above the cul-de-sacs for true hypertrophy of the supra-vaginal cervix, but this is rarely desirable, and requires much preliminary work, as in supra-vaginal amputation of the cervix for cancer.

Operations may be performed on the ligaments. We do not know how to shorten the broad ligaments, or the utero-sacral folds. Alexander's operation for the shortening of the round liga-

ments has not proved so satisfactory as it should do; and is now not often advocated as a permanent means of relief.

Ventro-fixation I need not say much about. It is an operation of expediency, not of necessity, and any mortality therefore is very regrettable; in spite of a few disastrous cases it is still not infrequently performed, and is justifiable in otherwise intractable cases. The abdomen is opened in central line, the uterus is pushed up by a sound into the wound, and is sutured there either to the peritoneum or to the muscle of the abdominal wall, as advocated by Dr. Herman. The most satisfactory operation of the kind is that of the late Dr. Keith. He brought up the uterus to the abdominal incision and drew out one of the ovaries and tubes and broad ligaments, cut off the ovary and tube on one side, and fixed the pedicle into the lower end of the abdominal incision. That deprived the patient of one ovary, but Keith gave a list of seventy cases with no death, so that his operation was justified as far as immediate results went, though sufficient time had not elapsed when he published those figures for judgment to be formed from other points of view.

The last operation I have to mention is vaginal fixation. I have done a few cases of this kind, and the method seems to bid fair to be very useful, particularly in bad cases of retroversion and prolapse with prolapsed ovaries. It consists of opening the vagina transversely in front of the cervix and anteriorly along its whole length from the anterior cul-de-sac to the urethra. The bladder is then stripped off from the uterus and vagina, pushing the mucous membrane back sideways. This requires a good deal of care. The bladder is thus turned up behind the pubes. Then the uterus is drawn down, and by means of a finger in the rectum or a sound *in utero*, the fundus is tilted forwards, and stitched to the anterior vaginal wall, passing sutures through the vaginal wall and through the posterior uterine wall in two or three places. The uterus is then in a state of anteversion, and if the vaginal outlet be also narrowed by a combined perinæorrhaphy and anterior colporrhaphy, a cure will result. If there be much mucous membrane, as in cystocele, it is advisable to remove some of it, so as to allow the parts to be drawn together without tension. Removal of the uterus for procidentia is probably never justifiable unless otherwise diseased.

**SOME EXPERIENCES
IN THE
SURGICAL OUT-PATIENT ROOM OF
A CHILDREN'S HOSPITAL.**

BY
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THERE enters first a pale worn-out woman, who tells you she is only twenty-two and looks at least ten years older. She brings you her infant, *aet.* 4 months. Since it was a fortnight old it has attended the medical out-patient room, suffering from the wind and chronic constipation, varied by sharp attacks of diarrhoea, eczema and chronic bronchitis. She says that she and her husband are quite worn out by the constant crying of the child. In desperation she occasionally yielded to the temptation and administered alcohol not only to herself but to her child. Nothing did this child more good. As an umbilical hernia has developed it is no longer regarded as medical, but has become surgical. The poor infant looks wasted and white; and as it commences to scream on your proceeding to examine it, the mother shoves into its mouth a rubber teat which is connected by a tube with a glass bottle, informing you at the same time that it is a very ravenous child and is never satisfied. The chest is small above, but below it is expanded by an enormously distended abdomen, which, when the child cries, bulges laterally and also along the middle line between the widely separated recti muscles.

In the position of the umbilicus is a thimble-shaped protrusion whose size varies with the explosive efforts of the child. The limbs are thin except about the joints, where the epiphyseal lines are abnormally prominent, and some of the long bones are bent. The head is large, the forehead prominent, and the area of the fontanelles disproportionately great.

You ask the mother what is in the glass bottle, and she tells you that it is a mixture of condensed milk and water, and that she uses condensed milk in preference to cow's milk as the latter readily becomes sour. She adds that she does not allow

the child to depend on that alone, as she found it did not satisfy it, but that she gives it one of the many foods which are freely advertised with the sanction of the State, in the lay and medical papers, as being good for young infants, and if she is not in sufficiently affluent circumstances to buy such expensive articles of diet she feeds it on Robb's biscuits, rusks, or even boiled bread.

She says she was never able to suckle this child, and attributes it to the fact that her strength had been undermined by three preceding pregnancies which followed one another in rapid succession. You inquire about these children, and you hear that two died of consumptive bowels a few months after birth, and the first was born dead after a tedious labour in which she was attended by a midwife of sorts.

Why should all this distress have been inflicted so unnecessarily and so cruelly on this little family, and who is responsible for it. These people are the victims of circumstances over which they have no control, and the State is alone at fault. She has certainly not neglected her child, but has attended to its every want with the self-sacrifice and unselfishness of a mother, which is, I think, one of the most admirable and wonderful qualities of nature. She has had to depend for her education in the diet and hygiene of her child upon information supplied by equally ignorant friends or relatives, and the very limited accommodation of her home renders it necessary that some means should be found to control the capacity of producing discomfort, which is possessed by an infant in a degree inversely proportionate to its age, with the disastrous results I have already related. Looking at the child you at once grasp the whole situation, and are able to consider the causation of the condition.

The child starts at its birth handicapped by an enfeebled vitality. What else could you expect from a young mother who has gone through the experiences this one has during her short married life. They are enough to break the spirit and undermine the health of the most robust. Not being able to supply the child with breast milk, she has had recourse to substitutes, with the awful results I have described to you. The frequent feeding produces a condition of discomfort, which is only relieved by the further introduction of food into the stomach. This produces constipation of

such a nature that the child is poisoned by its own excretions. This monotony is relieved by the diarrhoea which results from irritating food or faecal decomposition. The indigestion is associated with the production of much gas, which distends the abdomen, and is popularly described as "wind." The great increase of the intra-abdominal tension, brought about by its presence, interferes with the normal function of the abdominal wall in violent expulsive efforts as in coughing, so that mucus readily collects in the bronchial tubes, and the child suffers more or less severely from bronchitis. Again, this tension interferes with the action of the diaphragm, so that the supply of oxygen obtained is insufficient to keep the child vigorous and active, consequently it readily falls a victim to any such depressing influence as chill, or organisms obtaining a foothold in its tissues.

Besides over-distending, stretching and damaging the muscular wall, the tension results in a yielding of the weakest parts. At this age the umbilicus is the most yielding, and giving way, an umbilical hernia is produced. Associated with this enteric disturbance there exists in the peritoneal cavity a quantity of serous effusion. This may be present in sufficient quantity to give a distinct thrill on the abdomen being tapped with the fingers or, better still, by a lead-pencil held lightly in the hand. This fluid being exposed to the same tension is in infancy driven through the more or less pervious processus vaginalis, or into the canal of Nuck, so as to form the hydrocele of infancy with which we are so familiar; and bowel may also be driven along the same canal, or may be forced into an acquired hernial sac. Both these conditions you see remarkably well illustrated in the lad two years old who follows the last patient. He has bowel and fluid, in the left tunica vaginalis, while on the right side the tunica vaginalis and much of the processus vaginalis is distended with fluid. The communication with the abdominal cavity is very small, and considerable and prolonged pressure on the hydrocele is requisite to influence its size. The mother tells us that when he awakes in the morning it is very small indeed, and that it increases in size after he has stood for some time. On percussing the lower part of the abdomen with the handle of a pen while he is in the erect posture a very distinct thrill is felt by the hand placed on its wall.

Returning to the infant, we know that the bones have yielded because they are abnormally soft, a sufficient quantity of lime salts, etc., not being obtainable because of the condition of disordered digestion. Nature makes every effort to meet as far as possible this unsatisfactory state of affairs, and a quantity of imperfectly developed bone is deposited upon the original skeleton. This is most abundant where its presence is most urgently demanded, namely, upon the surface of the vault of the skull, in order to secure intact from injury the brain, the most important organ in the body to the life of the individual. On the back of this skull you find the bone less resistant than elsewhere, in fact it yields to the pressure of the finger. This results from an absence of deposit of new bone upon the skull in consequence of the pressure exerted on the head by the pillow, which in some cases is sufficient to produce an absorption or thinning of the normal skull, because of its imperfect nutrition.

The hurry to do its best to maintain the normal mechanical relationship of the individual to its surroundings is also very well illustrated by the rapid and abundant formation of imperfect bone at the epiphyseal lines, so producing the lumps in this position which are spoken of as beading. These are most conspicuous at the wrists, ankles, and costo-chondral junctions.

Now it is quite apparent to anyone that because the parents of the two children we have considered have not been taught how to feed their children they have brought upon them, and upon themselves, an immense amount of misery and distress. One feels inclined to blame them, but very little consideration at once shows us the utter injustice of such action, and that they are all the victims of a vicious system of government.

Another child comes into the room. She is about eight years old, and her mother complains that her right shoulder is growing out, and her ankles are weak. You inquire of the mother as to the number of her family, and she tells you that she has had nine children in rapid sequence, and that though they are all alive, they are feeble and delicate. She says that up to her marriage she lived in the country, where she was healthy and robust; but that her frequent pregnancies, anxiety about her children's health, limited accommodation, and very small means, had reduced her to the

wreck she now is. When asked if she takes any pleasure out of her children, she seems to regard them solely in the light of anxieties, and an infliction necessarily associated with matrimony. She has tried to make her elder daughters servants, and then factory girls, for both of which occupations they proved themselves physically unfitted. Now they earn a precarious livelihood as sempstresses. All her children suffer from weakness of the spine and ankles.

You look at the child, and notice that she is thin and frail, and that she occupies a listless position, supporting her weight almost entirely on the right leg. You examine her boots, and see that they are trodden down on the inner side, especially about the heel. They are pointed in the manner so attractive to members of the female sex as long as they pay any attention whatever to their appearance, and the worn-down heels are narrow, and so placed as to lie considerably anterior to the heel of the foot. This style of boot has produced in this child the form of foot which I have described to you as a typical female variety. The great toe is forced outwards, and the head of the first metatarsal bone appears to project inwards. The second toe occupies a form described as hammer-toe, being jammed into this position by the displacement outwards of the great toe and the inward displacement of the three outer ones. The high heel, placed as it is beneath the centre of the foot, has necessitated the transmission of almost all the weight of the body through the compressed and deformed anterior portion of the foot; and, as a natural consequence, the mechanically impaired foot readily wearies, and occupies the "position of rest" or of abduction, a posture described by the vulgar as "flat foot."

What can be more tiring or more wearying than wearing such instruments of torture? And how disinclined they must make the wearer to lead a vigorous active existence. Only women who can afford to drive everywhere can afford to wear them.

Unfortunately the poor mimic the rich, forgetting that their mechanical relationships to their surroundings differ very widely indeed. Do these people receive any instruction on such a simple, and yet how important, a thing as the physiology of the foot? No; there is too much of no

practical interest whatever that has got to be introduced into their brains to permit of time being utilized for such a valuable purpose.

As we already noticed, the patient possesses, in a condition of fixation, the resting posture which is assumed by the normal child when standing on the right leg; or, in other words, the "asymmetrical resting posture" of the trunk. It is usually described by the meaningless terms lateral curvature or scoliosis, which, like flat foot, have no connection with the condition, and do not in any way suggest its causation.

The asymmetry of the trunk is largely dependent on the depreciating influence exerted upon the feet by the boots, but both come about chiefly in a manner to which I will now call your attention.

Let us expose the thorax and abdomen, when you notice that the chest is flattened from before backwards, and that it does not appear to move during respiration. Pass a tape around the chest, at the level of the nipples, and you find that the variations of the measurements between inspiration and expiration do not exceed a sixteenth of an inch. Observe the abdomen, and you see that what breathing there is is of a most superficial nature, and is practically entirely diaphragmatic.

I would remind you of the subject matter of one of my anatomical lectures given last winter. In it I showed you that inspiration as carried on by the diaphragm is a more perfect reflex, and makes much less demand upon the capital energy of the organism than does inspiration produced by the intercostal muscles; that for this reason during our resting positions and during sleep the oxygenation of the blood and the removal of the carbonic acid from the lungs is carried on by the unaided action of the diaphragm; that old people whose chests like the rest of the skeleton have become fixed in symmetrical attitudes of rest depend almost if not entirely on the action of the diaphragm, and that feeble children with very little energy and vitality only obtain such an interchange of gases as results from the more or less regular contraction of this muscle.

I showed you that owing to the great differences in form that exist in the thorax in the two sexes, the diaphragm plays a much less important part in the female than in the male, and that a dependence on the diaphragm also is fraught with much more damage to the female child than to the male.

This you will readily verify if you examine the several children. Anatomists with wonderful obtuseness do not seem to have observed these important variations in the anatomy of the thorax in the sexes, with the most important points of physiological and hygienic interest which arise out of them, and even when they have been pointed out to them they do not seem to attach to them the importance they deserve.

They have discussed for a long time the mode of action of the intercostal muscles, and many fallacies have arisen in what is practically a very simple matter, owing to a want of recognition of physical conditions.

Attempts have been made to induce you to believe that there is any similarity in the mechanism of respiration and the influence exerted by rubber bands sloping in the direction of the intercostal muscles on two parallel bars which represent the ribs, which move on pivots upon two other parallel rods which represent the spine and sternum respectively, and as the result of this experiment the internal intercostal muscles are said to be expiratory, and the external inspiratory.

I will confine myself to criticising this particular experiment only. The fallacy involved in it consists in assuming that the movement of the ribs and sternum is the only change that takes place in respiration, and that the spine is a rigid rod.

Now I will show you that the variations in the curves of the spinal column in the positions of extreme inspiration and expiration are much greater than the variations in the movements of the ribs upon the vertebra, and that these movements are eventually dependent on one another. Observe a child in the active position of extreme inspiration, and you see that the dorsal spine is extended to its utmost, each vertebra moving upon that beneath it around a transverse axis, so that a vertical line dropped through that axis tends to get as far as possible behind in a vertical antero-posterior plane, a vertical dropped through that of the vertebra below. Look at the same subject in the passive position of extreme inspiration, and you find the dorsal spine is flexed to its utmost, each vertebra moving on that beneath it around a transverse axis, so that a vertical line dropped through that axis tends to get as far as possible in a vertical antero-posterior plane in front of a vertical dropped through that of the vertebra below.

The variations in the position of the dorsal vertebrae absolutely as well as relatively to one another, in a vertical antero-posterior plane, are very great indeed. Each pair of ribs attached to each vertebra follows its movement in the same plane, so that the point on one rib which lies immediately above that on the ribs immediately below in complete inspiration lies considerably anterior to it in complete expiration, the reverse being true in the transition from the position of expiration to that of inspiration. As the internal intercostal muscles slope downwards and backwards their points of attachment are nearest in extreme inspiration, while those of the external which slope downwards and forwards are nearest in extreme expiration. Therefore we see that the external intercostal muscles are muscles of expiration and most powerful flexors of the dorsal spine, while the internal intercostals are muscles of inspiration and the most powerful extensor muscles we have of the dorsal spine. All this I showed you was borne out in a most interesting and lucid manner by the examination of the bodies of labourers. I need hardly impress on you the importance of the knowledge that the internal intercostal muscles are by far the most powerful extensor muscles of the dorsal spine in the body, and that in the production of this extension of the spine they, by the associated movement of the ribs, increase the capacity of the thorax, and that the reverse is true also about the external intercostal muscles.

I would also point out to you that while the external intercostal muscles flex the spine and produce actively the position of the chest called extreme expiration, this position as one of rest is brought about solely by the inaction of the internal intercostal muscles. Therefore, during the assumption of this position the muscles and ribs exert no leverage action upon the vertebrae connected with them, nor oppose their rotating around an antero-posterior axis as they do when the body is supported on only one limb. The asymmetrical posture of rest of the trunk is associated of necessity with the position of rest of the thorax, namely, that of expiration, and it is impossible to obtain any rotation of the bodies of the vertebrae and the consequent existence of any lateral curvature of the spinal column when the chest is in a position of extreme inspiration, or,

in other words, when the spine is completely extended by the active contraction of the very powerful internal intercostal muscles. The knowledge of this important fact in the physiology of the normal skeleton supplies us with the clue to the measures we should adopt in the treatment of the fixed condition of the normal asymmetrical resting attitude of the trunk, which is commonly, and to my mind stupidly, described as lateral curvature, or, worse still, scoliosis.

The indications for treatment are as follows:

1. By making the child inspire forcibly to its utmost you exert upon the spine by means of the intercostal muscles and ribs such force as is exerted when the normal individual changes from the asymmetrical posture of rest of the trunk to the symmetrical active erect posture. Therefore, it is obvious that in attempting to overcome the fixation of the resting position we must adopt the same mechanism.

2. By making the child avoid the assumption of the asymmetrical posture of rest of the trunk. By causing the child to use for certain periods of the day the muscles of inspiration and expiration to their utmost capacity, not only do you bring into action the only mechanical arrangement we possess for restoring the resting dorsal spine to the symmetrical active position, but you supply it with a very much larger quantity of oxygen than it has been in the habit of obtaining, and you remove very thoroughly the carbonic acid. In fact, by ventilating the lungs you are supplying the tissues of the body generally with a large proportion of oxygen, an element of vital importance in enabling them to perform their functions normally. Beyond such attention to the respiratory function by means of which you supply the child with energy as capital, avoid at first making any great demand upon that capital as expenditure. I mean that you must not tire your patient by making her perform exercises other than those she is easily fitted to perform. The tissues in the most feeble can have their nutrition improved by massage without wearying. To cause your patient to make any demand upon its small capital by going through muscular exertion without attending to the supply of oxygen is practically to starve your patient still further than is already the case. There is no branch of surgery in which quackery and humbug are more rampant than in the treatment of this

asymmetrical resting posture, chiefly for the reason that the large majority of the members of our profession are unable to understand thoroughly the causation of the condition. Once they have grasped the physiology of the normal resting posture, they will learn to treat these cases themselves on sound scientific principles. I would not so much object to an unscientific treatment of the fixed normal resting posture, as good results in most cases, but I do decidedly object to cases of spinal caries being punished and hopelessly damaged by these means. Such cases are constantly coming under our observation.

This child presents another condition, of which you will see innumerable examples in the out-patient room. As you watch her breathe, you notice that her mouth remains open, and that the upper lip and teeth project forwards considerably below the level of the lower. In her ears you notice plugs of wool, and the mother tells you that earlier in life both ears discharged, and that at intervals when the child catches cold the discharge returns. On opening her mouth the palate is found to be very high, and the upper jaw narrowed very considerably in its transverse diameter. On inquiry you gather that this is a formation common to all the female members of this family. The mother tells you that the children suffered early in life from colds, which became chronic, and left them depreciated in hearing capacity, that several had suffered from inflammation and discharge from the ears, that they snored at night-time, and were greatly troubled with their tonsils. Two had had operations for adenoids, and one had had the tonsils excised. These operations had afforded some temporary relief in one case, but in the other inflammation and discharge from both ears followed immediately after, and a considerable discharge from the nose. The older girls, she says, have grown out of it, and are only troubled when they catch cold, when they become deaf. One cannot breathe through her nose at all, and at times suffers from a profuse watery discharge. She attended a throat hospital, where she had had a bone, presumably the inferior turbinated, cut out of each nostril. She breathed a little better for a time, but soon relapsed. She is now being treated with the electric cautery. The mother speaks disparagingly of the treatment, and says that by the time she recovers from the pain and aggravated

obstruction brought about by one application, it is time for her to undergo another, and she has to submit to the painful consequences over again. Her story, as she tells it, makes you wonder why she should come to you for advice, since her little family seems to have suffered much at the hands of surgeons. Surgery certainly does not seem to have impressed her very much, and she has all our sympathy. She and hers are victims of over-specialisation, but the chief sinner is again the State as represented by our miserably unsatisfactory and inefficient educational system.

Let us consider this last trouble, and examine into its causation and pathology. As I told you, the child—badly fed, both as regards the food put into its stomach as well as into its lungs—suffers constantly from the inroads of organisms, one evidence of which is so-called cold in the head. There are situated, as you know, in the roof and posterior wall of the pharynx a diffused mass of lymphatic glandular matter. This is covered by mucous membrane, which pockets into its substance. Again, in the faucial aperture are the tonsils, similar in structure but thicker and more circumscribed. Again, in the neck there are a number of lymphatic glands which receive much of their supply from these pharyngeal and faucial glandular collections.

Now a child gets a cold in its head. This means that the mucous membrane with the pharyngeal lymphatic tissue become swollen and inflamed, probably owing to the presence of organisms and their products. The mucus present in the nasal and naso-pharyngeal cavities assists in blocking the passage, so that air is unable to pass readily through it.

When the catarrhal condition subsides, the enlargement of the pharyngeal lymphatic tissue interferes with the passage of mucus from the nose into the pharynx, consequently it accumulates. Organisms grow in it, and it sets up further irritative change in the pharyngeal tonsil. It often runs out of the anterior nares, and irritates and inflames the orifice. The Eustachian apertures are often blocked by mucus being dammed back in the middle ear and antrum, and if the membrana tympani be already perforated, the discharge from the middle ear escapes into the meatus instead of into the pharynx. The occlusion of the orifice of the Eustachian tube frequently produces an acute

inflammation of the middle ear, followed by perforation of the membrane, and probably a chronic purulent otitis, or worse still, a more chronic inflammation, which leaves the membrane forcibly retracted, and the hearing capacity impaired to a degree usually largely in excess of that which exists in the chronic purulent variety, and without the same possibilities of improvement.

The faucial tonsils are frequently infected secondarily, and in the same manner. They become large, help to impede the passage of air through the only portal remaining. The lymphatic glands in the neck become swollen and inflamed, and if they do not subside very shortly, tubercular organisms too frequently find in them an area of depreciated vitality, in which they readily establish themselves, and from which they are often difficult to expel.

A considerable proportion of the cases of tubercular glands in the neck which abound in our out-patient room owe their origin primarily to a nasal catarrh. Since the enlargement of the pharyngeal lymphatic tissue has been observed by surgeons, it seems to have been regarded as the primary source of trouble in these cases, and a bloody and unpleasant operation for their partial removal has become very prevalent indeed. That this operation, like excision of the pharyngeal tonsils, is frequently of service one would not pretend to deny, especially in the case of poor people, who are unable to pay such attention to their children as is necessary, and who are unable to place them under favourable conditions and in very young children. The patient derives frequently a temporary alleviation from the operation, but in the majority of cases the cause not having been removed the effect again reappears. In not a small proportion of cases the patient suffers from instead of benefiting by the operation.

This treatment is obviously unscientific. Certainly in private practice it is in most cases quite unnecessary in my experience, and by itself comparatively useless. The obvious indication is to remove the source of irritation when the effects which result from it, if not already contaminated by tubercle, readily disappear. Establish a through passage for the air ventilating the nares and nasopharynx, removing the organisms and their products, and stimulating the mucous membrane from a condition of fulness, vascular relaxation, and

enlargement to its normal healthy condition. This can be done by patiently teaching the child to make such deep inspiratory and expiratory efforts through the nose, the mouth being closed as already described when considering the asymmetrical resting posture of the trunk. As the mucous membrane of the nasal cavities becomes more healthy, so the pharyngeal and faecal masses of lymphatic tissue regain their normal condition and size. The presence of the current of air through the nose and pharynx is a necessary factor not only to their development, but to the development of the upper, and to a corresponding though lesser degree of the lower jaw.

A CLINICAL LECTURE
ON
THE SURGICAL TREATMENT OF
TUBERCULOUS DISEASE OF
THE BLADDER.

Delivered at the Bristol General Hospital, June 10, 1896,*

BY
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IT is only within recent years that ulceration of the bladder, the result of tuberculous disease, has been dealt with by supra-pubic cystotomy. The first case published in this country was recorded by Mr. Battle, in the "Transactions of the Clinical Society"† for 1890. More recently Mr. Watson Cheyne‡ and Mr. Mansell Moullin§ have recorded cases, and Mr. Battle has published another case.|| Some French, German and American surgeons have also treated tuberculous ulceration of the bladder in this way, but the total number of cases published is not large. The operation has been undertaken when other measures, such as washing out the bladder with antiseptic solution, the injection of iodoform emulsion, and the application of lactic acid, have failed and the sufferings of the patient have been great. The ulcers have been scraped or cauterised, or both; and iodoform has

* The specimen of healed ulceration in the bladder was shown at the meeting of the Bristol Medico-Chirurgical Society, May, 1896.

† 1890, p. 207.

‡ "Lancet," June 22, 1895.

§ "Lancet," 1895, vol. i., p. 1308.

|| Ibid., vol. ii., p. 1165.

been rubbed in. In some cases the bladder has not been drained for more than a few days, whereas in others the drainage has been continued for weeks or months. Indeed, Mr. Watson Cheyne* and Dr. Pilcher, of Brooklyn,† believe that drainage is much more important than the direct treatment of the ulcers, for they point out that it would be very difficult, if not impossible, to scrape away all the tuberculous disease from the lax bladder wall. But several surgeons have supplemented the scraping by the use of the cautery, and Mr. Battle ‡ employed a solution of chloride of zinc, as the surface of ulceration was too extensive to cauterize. At the Chirurgical Society of Paris, Dr. Delagnière reported § a case in which the tuberculous portion of mucous membrane was dissected away through the supra-pubic opening, and the patient recovered. Baedenheuer has gone further than most surgeons and dissected away the entire mucous membrane.

In several cases the results of supra-pubic cystotomy, combined with direct treatment of the diseased mucous membrane, have been very good, relief has been quickly obtained, and generally the patients have made complete recoveries. In some the diagnosis of ulceration was made by the cystoscope; in others, in which the patients were females (as in the one the details of which I record) by digital examination; whereas in several the diagnosis rested on the symptoms and examination of the urine. Tubercle bacilli were found in many cases in the urine, but often were not discovered, though the tubercular nature of the disease was subsequently evident. Before describing my own case in detail I will refer to the more important points in some of the other recorded cases.

Mr. Battle's first patient|| was a girl aged 20. An extensive area of ulceration had been discovered by digital exploration of the bladder, and after supra-pubic cystotomy had been performed, this was scraped and a solution of chloride of zinc applied. The drainage-tube was removed from the bladder on the fourth day, and fluid ceased to flow through the supra-pubic opening by the fifth day. There had been symptoms of vesical disease for 16 months at the time the operation was under-

* Loc. cit.

† "New York Med. Record," 1892, vol. i., p. 256.

‡ Loc. cit., 1890. § "Medical Week," 1895, p. 162.

Loc. cit.

taken, and evidence of extensive ulceration for five months. Within two months of the operation she was free from pain, and there was very little pus in the urine, which she could retain for three hours. She completely recovered, and was quite well five years after the operation.

Mr. Moullin's* patient was also a woman, aged 22. The disease was of three years' duration, and the sufferings intense. The urine contained much blood and muco-pus. Ulceration was detected by exploration under anaesthesia, but the method employed is not stated. Supra-pubic cystotomy was performed, some of the ulcers were cauterised and others scraped. The drainage-tube was removed on the third day, and in three weeks the opening had closed, and a few weeks later all pain ceased, and the frequency of micturition passed off. Six months later the improvement was maintained, and she had gained flesh. There was then only a little mucus in the urine.

In Mr. Battle's† second case there was no ulceration in the mucous membrane of the bladder, but granulation-like projections were scraped away, and the bladder was drained. The patient completely recovered, but the case was published directly after, and sufficient time had not elapsed to enable Mr. Battle to say whether any fresh tuberculosis developed.

In Mr. Watson Cheyne's cases‡ the disease was with one exception limited to the bladder. They may be thus briefly summarised. In the first case the symptoms were very acute. Patches of intense congestion, with slight ulceration on their surface, were found in the trigone, and several miliary tubercles were seen in the mucous membrane near them. He completely recovered. In the second case there were the usual symptoms, and an ulcer was discovered by the cystoscope in the trigone, and a hard nodule felt *per rectum* in the base of the bladder. Drainage had to be continued for six months, but by that time the nodule had disappeared, and all symptoms had ceased. The third case was one in which, although the symptoms were of long duration, only a congested thickened patch and no actual ulceration was found in the bladder, but suppurative tuberculous disease subsequently developed in one kidney, necessitating nephrectomy. The boy recovered. The fourth

case was that of a man 49 years of age, and the lesion discovered was a thickened mass in the anterior wall of the bladder, from which a quantity of cheesy material was scraped away. No iodoform was used in this case. The bladder was drained for six weeks, and he quickly recovered. All these patients were under Mr. Watson Cheyne's care in the years 1891 to 1893.

Three successful cases have been recorded by Guyon* and one by Reverdin.† They were all males. One of Guyon's cases made a complete recovery, and was well four years after operation. In the other two, marked relief followed, but renal tuberculosis proved fatal one and two years later respectively. Dr. Pilcher's four cases‡ can hardly be taken as a group, and are not good test cases. In one, although the epididymis on both sides and the apex of one lung were affected with tubercle, the patient (a young man) completely recovered from the vesical tuberculosis after scraping the ulceration. The genital disease remained stationary, and the lung trouble cleared up. In another, in which the ulcerated area does not appear to have been scraped, but the bladder was filled with iodoform gauze for a week after the supra-pubic cystotomy, Dr. Pilcher does not consider that any material benefit resulted. The girl died of pulmonary tuberculosis. In the third, the disease was too general, and not sufficiently advanced in the bladder, to derive benefit. In the fourth case a diseased area of the mucous membrane of the bladder was scraped; the patient completely recovered, but there was considerable doubt as to the tuberculous nature of the disease.

Out of Dr. Bell's three cases, two were complicated with tuberculous diseases of the testicle, but these two almost completely recovered, and the uncomplicated case completely. In all the symptoms were severe, and ulceration was found in the trigone on opening the bladder. The ulcerated area was cauterized, and the bladder drained for some weeks. In one case the sinus did not close, but gave very little trouble. Watson of Boston|| found

* "Lecons Cliniques sur les Affections Chirurgicales de la Vessie."

† "Annales des Maladies des Organs Genito-urinaires," Jan., 1888.

‡ "N. Y. Med. Record," 1892, vol. i., p. 256.

§ "Journal of Cutaneous and Genito-Urinary Diseases," vol. x., 1892, p. 293.

|| "Boston Medical and Surgical Journal," 1895, vol. i., p. 123.

* Loc. cit.

† "Lancet," 1895, vol. ii., p. 1165. ‡ Loc. cit.

the operation of supra-pubic cystotomy with cutting the ulcers, and the local application of iodoform, partially successful when performed for the relief of the most distressing vesical tenesmus.

L. M., æt. 33, was admitted to the Bristol General Hospital on June 13th, 1895, with a history of eight months painful micturition. The pain began just before micturition and continued during the act, but did not last after its completion. There had been for several months marked increase in the frequency of micturition. She had been passing water every half-hour during the daytime, and several times at night. She said there had never been any blood in her urine; she had, in fact, noticed nothing abnormal about it. There was no history of pain in either renal region. She had lost flesh, but had not been coughing. From April 17th to the time of her admission she had been an out-patient under Dr. Parker, and during this time there had been pus in the urine in considerable quantity. This was so also on admission, pus cells and large epithelial cells, but no casts, being discovered on microscopical examination. The urine was acid. There was no renal or hypogastric swelling or tenderness. On July 4th, under anaesthesia, the bladder was sounded, but no stone discovered. The cystoscope also discovered nothing abnormal, although the vessels on the bladder-wall could be seen as clearly as the retinal vessels with the ophthalmoscope. I then very slowly dilated the urethra, first with my little finger and then with my index finger. With the latter I could feel almost every part of the bladder, and at the posterior surface at the base what felt like an ulcer with a calcareous edge was discovered.

The bladder was washed out with boracic lotion for a few days, but the process gave her so much pain it had to be discontinued. No tubercle bacilli could be discovered in the pus in the urine. With the exception of occasional rises to 100°, the temperature was normal. An attempt to drain the bladder, and thus keep it at rest by tying in a catheter, had to be given up as it increased her suffering so much. By October her condition had not improved; there was still a considerable amount of pus in the urine, which remained acid, and the pain in connection with micturition continued. Occasionally there had been some pain in the left loin, but there was no tenderness or swelling in either renal region. There had been

some irregular rises of temperature to 100° and 101°.

On October 10th supra-pubic cystotomy was performed, after irrigation with boracic acid lotion. The bladder was injected with the same fluid by means of an ovariotomy glass drainage-tube, around which overlapping layers of drainage tubing were placed, so as to form a conical plug for the urethra around the tube. It answered well both in this case and in another in which I tried it some years ago. The rectal bag, containing 8 oz., was used. On cutting through the abdominal aponeurosis and separating the recti, the fatty tissue which lies over the bladder came into view, with what looked like a fold of peritoneum lying over it. I could not feel the bladder easily, but on injecting more lotion it rose up under my finger quite unmistakably, and I pushed up the fold, tore through the fatty tissue, and opened the bladder; steadied on a sharp hook. By thoroughly drying the bladder by sponges in holders, and holding the edges of the opening well apart by the fingers and spatulae, I got an excellent view of the interior, and found extensive ulceration covering the left side of the posterior wall, and small white nodules scattered about in the mucous membrane in the same area. I scraped the ulcers with a sharp spoon, and rubbed in iodoform. I then united the incision in the bladder with catgut, around a large-sized drainage-tube, which was thus made to fit tightly, and supported the tube by uniting it with a couple of safety-pins to a cross-piece of tubing resting on the abdominal wall. The separated recti and divided aponeurosis were then united around the tube with catgut, and the skin with fishing-gut sutures. To the end of the tube in the bladder a glass tube bent at a right angle was attached, and from this a long india-rubber tube conveyed the urine into a receptacle. Nearly all the urine drained away through the tube, very little leaking out by the side into the cyanide and wood-wool dressing placed around, and none came from the urethra. She had no pain in the bladder or urethra for the first three days, but there was troublesome sickness on the 12th and 13th. On the 13th the tube slipped out of the bladder, and only a smaller one could be replaced, which allowed the urine to pass by its side into the dressings rather than along its lumen and into the receptacle. But later on Cath-

cart's suction apparatus* was employed with great advantage. It generally kept the bladder empty and the patient dry. The bladder was drained for thirteen weeks, and during this time, although there was a constant deposit of pus in it, the patient was as a rule free from pain, and when it did occur it was only a slight dragging pain, and not of the old character. She decidedly gained flesh during this time. The bladder was washed out with boracic lotion daily through the supra-pubic opening. After removal of the tubes the old pain on micturition in the region of the meatus returned to some extent, and she began to lose flesh again. There was no pain, tenderness, or swelling in the loins, and only occasional and slight rises in temperature. The supra-pubic sinus closed, and she left the hospital at the end of February, 1896. After her discharge the sinus opened, and urine escaped through it, but it soon closed again. She began to suffer from pain in the right loin, very severe at times, of an aching character, and lasting several hours, accompanied by nausea. These attacks often ended by the occurrence of a very sharp pain in the urethra and the passage of a calculus or calculi only a little larger than a millet-seed. She was readmitted on March 12th, and brought with her a dozen or more of these minute calculi she had passed. She located the seat of pain in the right renal region. The pain did not shoot along the ureter. There was tenderness in the right loin, but no swelling, and no tenderness or swelling in the left. There had been no pain in the left loin. She was quite free from vesical pain, but the urine containedropy muco-pus. No signs of tubercle could be discovered in the lungs, but she had lost flesh considerably. She remained free from any paroxysms of renal pain until April 1st, when one set in with great severity, and lasted until the 4th (though relieved by morphine), accompanied by constant vomiting. She died exhausted on the 7th. During the time of her readmission she had practically no rise of temperature. I was away at the time she died, but it was not thought wise to explore the right kidney, and the post-mortem shows that no good could have resulted had the operation been undertaken.

At the post-mortem examination the ulceration in the bladder was found to have quite healed, an

* "Brit. Med. Journ." 1895, vol. ii, p. 968.

area of scar tissue alone existing. The orifice of the right ureter was dilated, but just behind it lay a calculus which could not pass through into the bladder. The left kidney was riddled with caseous abscess cavities, but not enlarged. The right was considerably hypertrophied, and the pelvis and calyces contained some dozens of the same kind of minute calculi the patient had been passing, but not one as large as the stone found in the ureter. There were only a few minute tubercles in this kidney. The lungs were normal.

The case shows how advanced tuberculous disease may exist in one kidney without giving rise to pain, tenderness, or signs of enlargement of the organ. Had the disease not affected one kidney, or the other become the seat of calculus formation, there seems little doubt she would have recovered from the disease in the bladder; in fact, the post-mortem shows that she had recovered, inasmuch as all the ulcers had healed. Unfortunately, tuberculous disease of the bladder is often only a part of general genito-urinary infection, or may lead to it, but as Mr. Mansell Moullin points out in connection with his case, this more often occurs in men than in women. He thinks there is rather a tendency to exaggerate the frequency of tuberculous disease of the bladder being secondary to similar disease elsewhere in the genito-urinary track. Certainly some of the published cases referred to show that sometimes the disease is limited to the bladder, or at any rate the patients recover without symptoms of renal or genital tuberculosis. Even if we have evidence of tubercle elsewhere it has been suggested by Mr. Battle* that the operation should be performed as a palliative measure. In one of Dr. Pilcher's cases already referred to† it was quite successful, although tubercle was present in one lung and the epididymis on both sides; and in one of Dr. Bell's cases,‡ in which a tubercular testicle was removed, it was successful, though the sinus did not quite close.

I wish particularly to call attention to the value of Mr. Cathcart's apparatus for draining the bladder, which I used in this and another case. It keeps the bladder empty and the patient dry. A full account of it is given in the "Brit. Med. Journal," 1895, vol. ii, p. 968.

Sometimes the sinus left by supra-pubic drainage in cases of tuberculous disease of the bladder becomes infected with tubercle. A case of this kind is referred to in the "Clinical Journal," May 13th, 1896, p. 38. It occurred at St. Bartholomew's Hospital, and was brought forward at the Thursday consultation.

* "Trans. Clin. Society," 1890, p. 207.

† Loc. cit.

‡ Loc. cit.

THE CLINICAL JOURNAL.

WEDNESDAY, JULY 8, 1896.

A CLINICAL LECTURE ON THE OPERATIVE TREATMENT OF DISEASES OF THE HIP-JOINT IN CHILDREN.

Delivered at the Victoria Hospital for Children,
Tite Street, Chelsea, May 28, 1896, by

T. PICKERING PICK, F.R.C.S.Eng.

LADIES AND GENTLEMEN.—One of the objects for which this hospital was instituted was the “attainment and diffusion of knowledge with regard to the diseases of children,” and it was with the desire to carry out this object in its entirety that the medical staff have instituted the present course of lectures, in the hope that they may be able by this means to diffuse the knowledge of the diseases of children which they have attained in the wards and out-patient department of this hospital. Bearing this object in view, in selecting a subject for my lecture this afternoon, it seemed to me that I should best carry it out, and probably also best consult your wishes, by choosing some subject with which you were constantly coming in contact in your daily practice; rather than some uncommon and rare form of malady, with which perhaps you or I might meet with only one or two instances in our professional career. It was quite apparent to me, however, that I could not hope in the short space of time at my disposal to pass even briefly in review the etiology, the pathology, the symptoms and the treatment of disease of the hip-joint, and therefore I selected for our special consideration this afternoon simply the operative treatment which may be necessary or desirable in this affection. I did this because I have seen in my time great changes take place in the operative treatment of disease of the hip-joint, and I have seen that more can be done now than was thought possible to be done in the older days. These operative measures, except that of opening an abscess, were not under-

taken in the olden time, until the child was absolutely exhausted with the disease, and then undertaken only as a last resource. I can remember well when I was appointed to St. George’s Hospital that the teaching there was that the only justification the surgeon could bring forward for excising the hip-joint was the fact that the child was going down-hill, and that it would die unless an operation were performed. Frequently the children were in a condition of hectic fever or commencing lardaceous disease, and it was not until this had taken place that it was considered a right thing to operate. At this time probably the head of the femur was more or less destroyed, the acetabulum was perforated and matter was to be found in the pelvic cavity between the bone and the pelvic fascia, and the soft parts around the joint were perfectly riddled with sinuses. For the sake of comparison I will sketch to you very briefly the treatment of hip-joint disease in those days. I am not concerned to say very much on the treatment of the early stage; that was carried on very much in the same way as it is now. The child was kept at rest, generally with a long splint or extension with pulleys, and the same general attention was paid to health. The hygienic and dietetic conditions were carefully looked to. The treatment in those days was, however, more energetic than it is now. If a child’s leg was in an abnormal position, it was put under an anaesthetic and the parts brought into position, often with a very considerable amount of violence. This process was called by Bonnet “redressement brusque.” One constantly saw depletion in the shape of leeches; counter-irritation, especially the actual cautery and firm pressure by means of strapping and bandages resorted to in these cases. But I will take up the treatment of hip disease when the presence of an elastic swelling external to the joint denoted the formation of an abscess. The majority of surgeons left the abscess alone, and allowed it to burst. The abscesses could be watched getting daily larger and larger till eventually they burst, and then continued to discharge. Other surgeons opened the abscesses in various ways;

some opened them by a large free incision, others opened them by a puncture, others by a valvular incision, others by tapping with a trocar and cannula, others opened them under water, and others used setons, issues, &c.; but whatever plan was adopted the result was always the same,—the abscess continued to discharge, a sinus was left and the pus became septic, matter burrowed in the soft parts, which became riddled with sinuses, and the child ultimately began to sink, exhausted with hectic fever or lardaceous disease.

Then, with the child in this condition, and then only, did the surgeons venture to suggest excision of the hip. No wonder that the results were so disastrous. In 1868 Holmes published his statistics to justify the operation of excision of the head of the femur. He operated in all on nineteen cases. Of these, six died from the direct effects of the operation; one died after the operation from previous effects of the disease; one died after amputation; two recovered from the operation but died of disease; two were not benefited by the operation; one case was doubtful; there were three who had useful limbs but with discharging sinuses, and three only of the nineteen cases recovered: we may say of these nineteen, then, that only three were successful cases; for as regards the three with useful limbs and sinuses, I think it is evident that these cases would terminate fatally at no far distant period of time, either through lardaceous disease or dissemination of tubercle in other organs.

About a decade later, Mr. Croft struck what was to my mind the keynote of the more successful treatment. In December, 1879, he read a paper before the Clinical Society of London, on forty-five cases in which he had performed excision, and he endeavoured to show that greater success attended excision if done before the abscess had been opened. I remember the reading of that paper very well, and I remember the discussion that followed, and I also have a clear recollection of the feeling which was evidently present in the minds of most of the speakers, that the operations of Mr. Croft had been a little too bold, and that probably many of the operations had been performed unnecessarily, a feeling which I must confess I at that time shared, though Mr. Croft was very careful to point out that before he had performed any of the operations, all other known treat-

ment for the arrest of the disease had been tried and had failed. Mr. Croft formulated three rules in regard to excision and the treatment of hip-joint disease. First, when there is a collection of fluid in and about the joint in a case of well-marked hip disease, especially if associated with starting pains, an antiseptic incision should be made as if the surgeon intended to excise, and he should only desist on finding the articular structures in a condition from which they could rapidly recover and yield a movable joint. Second, when pus associated with pan-arthritis (or strumous disease of joint in children) is known to be present, even if the surgeon is uncertain with regard to the state of the bone, the surgeon should excise. Third, if the surgeon is certain that necrosis has occurred, he should certainly excise.

My reason for giving this brief history of the teaching with regard to the operation of excision of the hip, is first because it leads us to the starting-point from which, in my mind, operative interference is necessary, and second, because it has enabled me to quote Mr. Croft's conclusion, and it will be my endeavour to show in this lecture in what particulars I agree in these deductions of Mr. Croft, and in what points I differ from him.

There is, I suppose, no surgeon nowadays who would deny that as soon as an abscess is formed, the time has arrived for operative interference. And it must be understood that in using the word abscess, which is perhaps not a very correct one, but at the same time a convenient one, I mean to include all those cases where the tuberculous material has casedated and broken down, and formed a curdy fluid, no doubt in most instances mixed with pus from surrounding inflamed tissues.

When this has taken place, there is no prospect of any amelioration except by the evacuation of the curdy material which has formed. But we should be very sure that there is pus or casedated tuberculous material before operating. I do not agree with Mr. Croft in thinking that it is necessary to incise every swelling in hip-joint disease. I have seen many cases with swelling at the hip-joint in which the swelling has existed and remained even for months, and then finally has disappeared and the child has recovered without any operation or incision being necessary. Therefore make sure that matter is present, and there is an easy way of making sure by using the exploring syringe. There

is a little girl upstairs on whom I propose to do this to-morrow; there is some swelling and there is an old history of hip-joint disease. I intend to satisfy myself to-morrow by using the exploring syringe whether there is matter in the joint or not. There is one word of caution: you should always use a needle of large size. If you use a smaller needle it may become blocked and thus you may get no result, and the exploration would be positively harmful because it would mislead. We start then from this point. Given a case in which there is undoubted evidence of the presence of an abscess we proceed to operation, that is to say, we open the abscess and deal with the condition that we find.

The first question which presents itself to us is the best place in which to make the incision for this purpose. I can have no doubt in my mind that in the majority of cases the best place to make the incision is in the line which was advocated by Hueter of Greifswald, and by Parker of London, by what is known as the anterior incision, not so much on the grounds given by Parker,—because it avoids the necessity of cutting through the gluteal muscles attached to the great trochanter,—but because it gives us an easy access to the joint and the neck of the bone and enables us to deal with any condition which may be found, and therefore in those cases where I have to open an abscess I always make the incision from a little external and below the anterior superior spine of the ilium, in a direction downwards and inwards in an oblique direction, cutting between the sartorius and the tensor vaginæ femoris, and then between the rectus and the glutei. By this means the neck of the femur and the capsule of the joint can be easily reached and the puriform fluid evacuated. This latter should be done as rapidly as possible and the abscess cavity scraped and well sluiced out with hot sterilized water or antiseptic solution, so as to get rid of all cased material as quickly as may be, so as to prevent any more contamination of the wound than is absolutely necessary. A careful exploration should now be made and the exact condition of things ascertained as far as possible. The first thing is to ascertain whether the disease began in the synovial membrane or in the bone, for I strongly hold to the opinion that the disease in the hip-joint may begin in either of these situations.

I am aware that many surgeons think that hip-joint disease invariably begins in the bone, but I have reason to believe that it is not so, and that it may begin in other places. I have collected sixty-four cases of excision of the hip-joint which have occurred under my care or under the care of my colleagues in which I have examined the parts, and of these sixty-four I believe the disease began in eleven cases in the synovial membrane, and in the other fifty-three in the bones. In coming to this conclusion I have been guided by the appearances of the parts after removal, selecting that part where the disease was most advanced as the probable origin, and therefore my deductions may in some cases be open to error, but I think they represent very fairly as a whole the actual state of things. In order to ascertain the condition of the head of the bone, the tract by which the abscess has escaped from the joint must be sufficiently enlarged to allow the introduction of the finger. In a few cases, on introducing the finger the bone will be found to be quite hard and firm, though denuded of cartilage, and on passing a probe it will be found to impinge on hard bone into which it cannot be buried. These, I assume, are cases where the disease has begun in the synovial membrane, and under these circumstances I do not remove the head of the bone, but I do what I may call a limited erosion: I scrape away as far as I can all the tuberculous tissue and diseased synovial membrane and flakes of cartilage which remain on the surface of the bones. I then wash out the joint with hot sterilized water or some antiseptic lotion, generally using corrosive sublimate, introduce a drain into the joint, inject some iodoform emulsion, and sew up the wound. I do not do what is recommended by some, turn the head of the bone out of the socket and scrape it and then replace it. Subsequent treatment consists in daily flushing, and the fluid which I use is iodine—a drachm of the tincture in a pint of hot water. The joint is thoroughly flushed out with this, and the limb is kept perfectly at rest on a double Thomas's splint. If the discharge continues for six weeks and shows no prospect of becoming thinner or less in quantity, then under these circumstances I give up the case as hopeless, and I at once proceed to excise the joint. But if, on

the other hand, the discharge becomes less in quantity and thinner in quality, then I persevere with this line of treatment, and in some cases I secure a bony ankylosis between the head of the bone and acetabular cavity, and a better limb than I should have got by excision. I am bound to confess, however, that in the large majority of the cases this fails, certainly in fifty per cent, but my argument is that the procedure does no harm and that if it succeeds you get a much better limb than you would get if you had excised the head of the bone. I do not think that, in any case, the experiment has ever done any harm, unless a second abscess forms, and then the case is not quite so favourable for a future excision; but if you are very careful with the drainage this does not occur. I should like to quote a case which occurred to me last year in connection with this plan of treating these cases.

I was asked to see a little girl, æt. 7, in July of last year, just before I went away for my holiday. She was suffering from well-marked tuberculous disease of the hip, with a fluctuating swelling over the back of the joint. The history which I obtained was that her father had died of phthisis. Early in last year she was noticed to complain of pain in the hip, and to limp in walking. A medical man was consulted and diagnosed hip-joint disease, and she had been treated ever since by rest in bed, with extension by weight and pulley.

She was a well-nourished child, and presented no other signs of tuberculous disease. The swelling was about the size of a Tangerine orange, and was situated directly over the back of the joint, beneath the glutei muscles. The sense of fluctuation on account of the depth of the swelling and the overlying muscles was not very definite, and it was not known how long it had existed. In fact, the swelling had not been discovered previous to my visit. I determined first of all to aspirate the swelling, partly to make sure of the presence of matter. This I accordingly did, and drew off about an ounce or an ounce and a half of curdy pus. As I was leaving town in a day or two, and the mother was anxious to take the child to the seaside for a time, it was determined that nothing more should be done until my return.

I saw her again in September, and the child's condition was then materially worse. There was a large fluctuating swelling reaching halfway

down the thigh, and involving the whole of the buttock. The child was in great pain, and could not bear the slightest movement of the limb. She looked wan and anxious, and the temperature was 101° F. A free incision was first made into the abscess over the back of the joint, and about half a pint of pus evacuated: a second opening was made at the most dependent point of the abscess, about the middle of the thigh, and the whole abscess cavity carefully scraped out and flushed with hot boracic solution, and well scrubbed with dry gauze. Attention was now directed to the joint. The hole in the capsular ligament through which the pus had tracked was enlarged, and the finger introduced into the joint. The head of the bone and the acetabular cavity as far as they could be felt with the finger were entirely denuded of cartilage, but the bone felt fairly hard and healthy, except in one spot where it was a little softened. This part was carefully scraped with a sharp spoon. The interior of the joint was then scraped and rubbed with dry gauze in every part which could be reached by turning the limb in different positions, and well irrigated. Drainage-tubes were introduced down to the joint, and the rest of the wound sewn up. Some iodoform emulsion was injected, and the parts dressed. For some time after the operation there was a free discharge, with a nightly rise of temperature and some starting pains at night. The wounds were well irrigated daily with iodine solution. After a week or two the discharge began to lessen and become thinner. The lower tube was gradually withdrawn, and the abscess in the thigh completely closed, and there was only a little discharge from the upper tube, which passed directly down to the joint. Her general condition manifestly improved, she lost her pain, and her temperature became normal. About the beginning of December the discharge practically ceased; the tube was withdrawn, the wound closed, and she was allowed to get up and go about on crutches in a Thomas's splint, with a cork sole on the other boot. She is still going about on her crutches, but she is quite well, and there is firm ankylosis of the bones in a good position.

Now I think that no one can doubt that this child is better than if she had had an excision of the head of the femur; there is firm ankylosis, probably bony, in a good position. It is true that this is an exceptionally good case. I must, how-

ever, be fair and allude to the other side of the question, and I shall have the pleasure of showing you a little girl whose hip I excised a month ago, and I will give you the notes of the case.

Maude F., æt. 3½, was admitted in August last with the history of symptoms of disease of the hip of nine months' duration, during the greater part of which time she had been treated with a Thomas's splint. There was a strong history of phthisis in her family. When admitted, there were the usual symptoms of hip-joint disease, and a fluctuating swelling on the front and outer side of the joint. I cut down on this swelling, and opened an abscess which communicated with the cavity of the joint. The whole of the cartilage had disappeared from the head of the femur, but the head of the bone did not seem unduly softened, and there was no thickening about the neck of the bone. I therefore carefully scraped away all the granulation tissue which I could reach, and thoroughly irrigated the joint, introduced a drainage tube, and sewed up the rest of the wound and dressed it in the ordinary way. For a time things went on very well, and there was a minimum amount of discharge, so that she only required dressing every third or fourth day, and there was no rise of temperature; but the wound did not heal. Accordingly, on December 13th, I explored the wound, but still found little or no evidence of any disease in the bone, and therefore I determined to give it another trial, and having again scraped out the sinus I redressed the wound. Unfortunately, shortly after this she was attacked with diphtheria, and had to be removed to the fever hospital. She was readmitted at the end of March, and upon examination it was now found that the head of the bone was extensively diseased; it was roughened and eroded on its surface, and so soft that a probe could be passed into it without any difficulty. Excision was therefore performed on April 10th. She is now progressing favourably, though there is still a discharging sinus.

We now pass on to consider quite a different class of case. We now consider those where the disease of the hip started in bone, and these, as we have seen, constitute the greater portion of the cases with which we have to deal. The disease may begin in four different situations. By far the most common place for it to begin is in the ossifying tissue of the diaphysis in contact with

the epiphyseal cartilage; but it may also begin in the centre of the cartilaginous epiphysis of the bone, or in the ossifying tissue of the trochanter or in the acetabulum. In the sixty-four cases to which I have already alluded, and of which fifty-three began in the bone, forty-four began in the head or neck of the femur, five in the acetabulum, and four in the trochanter. I have been obliged to classify together the cases in which the disease began in either the head or the neck, for it was impossible from the examination of the specimen to distinguish in which of the two places it commenced. I wish, however, to confine my remarks to those cases where the disease begins in the growing tissues at the end of the shaft of the bone at the under surface of the epiphyseal cartilage. It is foreign to the scope of this lecture to enter into the pathology of the subject, and it will be sufficient for my purpose to state that probably owing to some strain or wrench a limited rarefying osteitis is set up in this situation. This inflamed tissue is invaded by the tubercle bacillus, a deposit of tubercle takes place, surrounded by a large mass of granulation tissue, and the ordinary tissue becomes destroyed and replaced by the new material. This inflammatory process spreads along the soft vascular tissue on the under surface of the epiphyseal cartilage, either in one direction, until it reaches the surface of the bone, or it may burrow in all directions, so as to separate the bone from the shaft. When the inflammatory process reaches the surface of the bones, it usually spreads to the synovial membrane, and the whole of the structures of the joint become involved and a general arthritis sets up.

But it may happen in some cases that as the inflammatory process in the bone spreads outwards in one direction, adhesions take place between the layers of synovial membrane covering the neck of the bone and the capsular ligament, and the caseated material does not find its way into the joint, but extends through the capsular ligament, and an abscess forms in the structures outside the joint without involving its general cavity. This was so in the two cases from which these drawings were made.

Now it may happen when one opens an abscess in connection with a case of hip-joint disease, that after flushing it out, evidence may be found that

the disease has commenced in the position we have been discussing. To take the severest cases first, we may find that the abscess has spread in all directions and that the shaft is separated, and the head of the femur lying loose in the cavity; then, of course, you must excise. But in the majority, perhaps, of cases we find on the front of the neck of the bone a little exposed spot, and here we find a minute perforation; on introducing a probe it will enter a cavity in the neck of the bone. This was the case with a little girl at present in the wards of the Hospital, whom I shall show you presently.

Beatrice H., æt. 7, was admitted into the Hospital at the latter end of last year with caries of the cervical spine and tuberculous disease of the knee. Whilst in the hospital she developed symptoms of morbus coxae, and after a time a fluctuating swelling appeared on the front and outer side of the upper part of the thigh. On February 25th of this year I cut down on the abscess by the anterior incision, and having opened it, I examined the neck of the bone and found a little exposed spot on the front of the neck, in which was a minute opening. Upon gouging the front of the neck of the bone away, I opened up a cavity in the shaft of the bone, containing pus and carious bone. This was carefully scraped away. I could not satisfy myself in this case that the joint was implicated, for there was no swelling of the capsule, and the movements of the articulation were perfectly smooth. It was therefore not interfered with. The parts were irrigated and cleaned up. A counter opening was made on the outer side of the joint for draining, and the wound dressed. As you will see after lecture, she has gone on very well since the operation, considering the numerous foci of tuberculous disease which she presents. The wound is entirely healed, and in a week or two she will go down to our Convalescent Branch at Broadstairs, and I trust that she may entirely recover.

But in the great majority of cases where the disease has arrived at the stage of formation of abscess external to the bone, that is to say, the stage at which operative interferences is undertaken, it will be found that the joint is implicated and is full of pus. In these cases, in spite of the implication of the joint, I am sometimes disposed

to attempt to save the head of the bone, provided there is no evidence of the disease having extended itself to this structure, that is to say, in those cases where the head of the bone is smooth and hard and not in any way eroded. The following may be quoted as an illustration of this class of case:—

Daisy T., æt 5, was admitted under my care on September 17th of last year. The history obtained was that the disease had existed for about a year. During the latter part of 1894 she had been an in-patient at the Children's Hospital, Great Ormond Street, and had then been discharged and was treated at home with rest, but without extension, for six months. She was then placed in a double Thomas's splint. Shortly before admission a swelling had appeared on the outer side of the thigh.

When admitted, the child was in good condition, but there was a large fluctuating swelling at the front and outer side of the upper part of the thigh.

On September 24th the abscess was freely opened, scraped and flushed. A pin-hole opening was found in the front of the neck of the bone; this was enlarged and an abscess cavity in the neck of the bone opened up and scraped. The joint was full of pus; the front of the capsule was therefore freely incised. The cartilage over the head of the bone was found to be eroded in patches, but was not destroyed over the greater part of the bone. The joint was scraped and freely flushed. A drainage-tube was inserted into it and brought out at a counter opening on the outer side of the thigh at the most dependent part of the abscess. The first wound was sown up in its entirety.

She went on well after the operation, the discharge quickly lessened and soon consisted of a drop or two of semi-purulent fluid in the twenty-four hours. This after a time ceased, and the notes on the 13th December state that the sinus was healed. Unfortunately, on December 22nd a case of scarlet fever occurred in the ward, and she had to be sent home and has not since been heard of, nor have I been able to find her.

There is another plan of dealing with these cases of disease commencing in the neck of the bone, to which I must briefly allude, though it is unfortunately a plan of treatment which can rarely be adopted, on account of the obscurity of the

symptoms of the disease in its early stage before the abscess has perforated the bone and found its way into the soft parts, the only stage to which this plan of treatment is applicable. I allude to the mode of treatment first advocated by Sir Thornley Stokes of tunnelling the great trochanter and femoral neck ; though he does not appear to have appreciated the real motive of the operation, as he states that his object was to open up the cancellous tissue of the neck, and thus relieve tension, whereas the real object is to reach a focus of suppuration in the neighbourhood of the epiphysial cartilage and by scraping it out and removing all the tuberculous material to prevent contamination of the joint. The operation is performed by making a vertical incision over the trochanter, and applying a small (half-inch) trephine to its outer surface, and carrying it in the direction of the head of the bone to its full extent. I have performed this operation, and though, as subsequent events proved, I was right in my diagnosis as to presence of a suppurating focus in the neck of the bone, I failed to reach it, though the bottom of the tunnel was explored in all directions with a small gouge. I am not inclined, therefore, to repeat this mode of proceeding, especially as we have a much easier plan of reaching the neck of the bone by the anterior incision to which I have alluded.

We now pass on to consider what after all is the plan of treatment which will have to be followed in the majority of cases of hip-joint disease, where suppuration has taken place, namely, excision of the joint. For in most cases when the abscess is opened and the parts examined there will be found to be such evidence of disease in the bone as will make it perfectly clear to the operator that the only way of bringing about a successful issue is to remove the head of the bone.

There are several ways of doing this : one way is by the posterior incision through the glutei muscle,—this was the old plan ; then, secondly, there is the plan by the external incision ; and thirdly, the plan by the anterior incision, to which I have already alluded. The second plan, by the external incision, was in vogue twenty years ago, when surgeons were inclined to advocate a much more extensive removal of bone than is usually adopted in the present day. Of these three plans I give decided preference to the anterior

incision, in the first place because it is the most convenient for exploration, and having made the opening to explore, if it is found desirable to continue the operation, it is not necessary to make another incision ; and then another advantage of this anterior incision is that no structure of any importance is cut through. The only objection which has been urged to this operation is that it does not afford very efficient drainage, but if the plan of proceeding is carried out on the lines laid down by Mr. A. E. Barker, which I shall describe immediately, no drainage is required.

What I have said about the external incision suggests a word or two about the amount of bone to be removed. No more bone ought to be removed than is absolutely necessary ; if the section is made below the trochanter it is quite clear that the primary shortening must be greatly increased. But this is not all ; in those cases where this large amount of bone has been removed, the bond of union will be loose and long. So that as soon as the child begins to get about the weight of the body will stretch the union and thus very considerably more shortening will take place and a very unstable limb result. What we should aim at is to secure as good a bond of union as is possible. I do not suppose that bony union ever occurs, though this would be the best, but what we should aim at is a very strong fibrous union. If the bone is cut through below the trochanter this is impossible, but if the neck of the bone has been sawn through at about its centre, the remains of the neck can be forced into the acetabulum by abduction of the limb, and thus the two exposed surfaces of the bone can be brought closely together and firm fibrous union will be the result. With regard to the way of performing the operation, I follow rigidly the plan laid down by Mr. Barker, which, to my mind, is most excellent and leads to the most satisfactory results. The abscess having been opened in the manner I have indicated, the parts are flushed out so as to get rid of all the tuberculous material as quickly as possible, and then any structures round the joint are cleared with a scalpel, and the neck sawn through with an Adams' saw, and the head removed with a pair of sequestrum forceps. The acetabulum must now be examined with the finger to ascertain whether it is involved in the disease

and to what extent. It and the whole abscess cavity must be thoroughly scraped until every particle of diseased tissue is got rid of. In doing this the most useful instrument will be found to be Barker's flushing gouge, which washes away the débris as fast as it is separated. When the cavity is cleaned it should be dried and a sponge introduced, stitches are then inserted through the edge of the wound, but these are not tied at once. As soon as everything is ready the sponge is removed, iodoform emulsion is introduced into the cavity, and is allowed to remain there for a minute or two, and is then pressed out by the hands of an assistant while the stitches are tied. The limb is then abducted, and in this position the wound is dressed. The whole limb is put up in plaster of Paris or Thomas's splint, or arranged with sand-bags. These cases do not as a rule require dressing for ten days, provided the temperature remains normal; the wound is then dressed, and the stitches removed, and that is all that is necessary. The child is, however, to be kept in bed on a double Thomas's splint for three months with the limb in a position of abduction. If the weather is warm it can of course be carried out, but it must be kept flat on its back. After this it may be allowed to use a single Thomas's splint. I make it a rule never to allow a child to put the excised limb to the ground for twelve months after operation, so as to ensure a firm union.

In conclusion, I must say a word or two upon the operative treatment of quite a different class of cases to those which we have been considering. Occasionally it will happen that we are consulted on cases of hip-joint disease that are more advanced than those I have been talking about, with extensive caries and putrid sinuses. This as you will remember was the condition in which excision was performed in former days, when 60 per cent. of deaths occurred. I am strongly of opinion that no excision should be done in these advanced cases. You cannot remove the disease, and any operation is useless, and you will get a better result by strict cleanliness, thorough drainage, and sending the patient to the seaside. But the child should be zealously watched, and if it shows indications of going down hill, or any symptoms of commencing lardaceous disease of the viscera, amputation at the hip-joint should be immediately performed. I have seen many lives saved by

amputation in these advanced cases, though of course at the expense of the limb. But it is an operation which should not be undertaken without the gravest consideration, and solely as a last resource, to save the life of the child; for it is a most serious operation as regards its immediate danger, especially in young children, and those who are worn out by long-standing disease and protracted suppuration. And, moreover, it must be borne in mind that the operation, even if successful, very seriously mutilates the patient, and condemns him to a life-long progression on crutches, for as far as I know no artificial appliance has ever been made which has proved efficient as a means of support and progression.

A CLINICAL LECTURE ON TYPES OF PULMONARY TUBER- CULOSIS.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton, on 3rd June, 1896,

By F. J. WETHERED, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

THE word "phthisis," as indicating a wasting disease characterised by certain morbid changes in the lungs, is rapidly disappearing from medical literature, and the more scientific term "pulmonary tuberculosis" is taking its place. I have taken as the title of my remarks this afternoon, "Types of pulmonary tuberculosis," or, if you prefer the old nosology, "Types of phthisis," and I thought it would be more in accordance with the wishes of my hearers if I considered it mainly from a clinical standpoint, although I shall have to refer to the morbid anatomy to more fully explain the classification I have adopted. I shall endeavour to make this lecture as far as possible a demonstration, bringing before your notice patients who exhibit the various types. I say "as far as possible" advisedly, for there are at least two forms that are so uncommon, or of such a nature, as to be rarely seen in this hospital.

The classification I shall follow is in a great

measure taken from Dr. Douglas Powell's book on "Diseases of the Lungs," for I believe it is the most practical yet introduced.

ACUTE PULMONARY TUBERCULOSIS :

Disseminated: Miliary Tuberculosis,
Florid phthisis.

Confluent form.—Acute pneumonic phthisis.

SUB-ACUTE AND CHRONIC PULMONARY TUBERCULOSIS :

Chronic pulmonary tuberculisation.

Chronic catarrhal tuberculosis of the lungs.

Fibroid tuberculosis of the lungs.

This is the main classification. In addition I propose to show you clinical types of—

Incipient pulmonary tuberculosis.

Advancing " "

Arrested " "

Basic " "

Further, I shall refer briefly to other clinical types—

Tubercle engrafted on bronchiectasis.

Tubercle engrafted on emphysema.

Pulmonary tuberculosis arising in the course of diabetes.

Chronic fibroid tuberculosis originating in dusty occupations (dusty phthisis).

The adjectives "catarrhal" and "fibroid" are used to designate the predominant element of the type, and do not refer in any way to the ætiology of the disease.

Acute pulmonary tuberculosis :

Disseminated: Miliary tuberculosis.

This form is distinguished by the rapid evolution of a large number of grey or yellow granulations in the lungs, and commonly also in other organs of the body simultaneously with the lungs. The disease in adults is comparatively rare as compared with the ordinary varieties of chronic or acute pulmonary tuberculosis, and in a considerable proportion of cases it appears to originate in the rapid dissemination of tubercle, secondary to that already existing in the lungs or elsewhere. In a certain proportion, however, it commences as a primary disease, and is then probably due either to a strong predisposition, or to excessive insanitary conditions, though in some its origin remains unexplained. Experimental pathology suggests that its rapid extension and dissemina-

tion are in some respects proportionate to the amount of poison introduced into the system.

In its pure form it is seldom met with in this hospital, although we occasionally find miliary tubercles scattered through the whole of the lungs at post-mortem in cases where there is advanced disease of the upper lobes; but I do not include these cases under this heading. At one of the London hospitals I had occasion to meet with several marked cases of miliary tuberculosis due, I thought, to defective ventilation and disposal of the sputum; but of course it is very difficult in these cases to distinguish between *post hoc* and *propter hoc*. Still when structural alterations were made and disinfectants were used in the spittoons, the cases lessened in number although they still occurred.

Cases of miliary tuberculosis of the lungs are seen chiefly at general hospitals especially in children, occurring with acute disseminated tubercle in other organs.

The purely acute disseminated tubercular affections run their course to a fatal issue with little or no softening, and, during life, there is often considerable difficulty in making a diagnosis between such an affection and typhoid fever. I well remember a case which occurred at the Middlesex Hospital, under the care of Dr. Douglas Powell, in which such a difficulty arose. I examined the sputum for nineteen days in succession and failed to find tubercle bacilli. I found them on the twentieth examination, but failed again on the twenty-first. On the death of the patient very soon afterwards, the lungs were found studded with miliary tubercles. I am unable to show you such a case to-day clinically, but pathological specimens are on the table.

Florid phthisis—"galloping consumption." In this form tuberculosis commences simultaneously in both lungs and runs a very rapid course. It usually occurs in young adults, generally females, who are often brunettes, with sharply marked features, long eyelashes and fine skins, with delicately marked veins. When attacked, the flushed face, the bright eyes and alert mind filled with hopefulness, and little knowing the terrible disease with which they are affected, contrast strongly with the apathy, pallor, and prostration of the other forms of acute and chronic tubercle.

A case which came to my out-patient room was

that of a woman aged 21, who had only been married three weeks, having been quite well until six weeks before marriage, when she was attacked with what she called pneumonia, but rallying a little the marriage ceremony was performed. She was of the type I have just described. Foci of breaking-down lung tissue were easily appreciable by the physical signs on either side; dyspnoea was extreme. I advised her early admission. Whilst in the hospital her temperature was hectic in type and the course of the disease was rapid; the prognosis being hopeless, she was removed at the husband's request. I heard no more of the case, but death probably occurred in three or four weeks. The patient rarely lives more than four weeks to three months after the commencement of the disease. In most cases, post-mortem the lungs are found to present numerous areas of greyish-pink granular consolidations with yellow caseous centres broken down into small cavities communicating frequently with enlarged, more or less eroded, and visibly inflamed bronchial tubes. No miliary granulations of tubercle are to be seen, although the small yellow centres may at first sight resemble them.

Confluent form.—This form is also known as "acute pneumonic phthisis," or, as the Germans aptly call it, "tubercular caseous pneumonia." The disease is usually one-sided in the initial attack; the consolidation is extensive and very dense, the disease being most massed in the affected lobe, occupying perhaps its entire extent. This form is very apt to be confounded with croupous pneumonia of the apex; it very rarely occurs at the base of the lung. The signs of consolidation are uniform over an extensive area, bronchial breath sounds and crepitant râles, at first fine, then larger and more liquid are heard, constituting what are known as "humid clicks." These clicks are one of the most characteristic signs of disintegrating lung-tissue. There is also pectoriloquy. The onset of the disease is abrupt and stormy, with pain in the chest, short cough and scanty expectoration; sometimes haemoptysis, with raised temperature, hectic in type, rapid pulse and frequent respirations. Although the prognosis is as a rule bad, provided the disease does not extend to the opposite lung, recovery often occurs by elimination of the caseous products, cicatricial consolidation of the cavities thus formed, and

compensatory development of the opposite lung. Formation of cavities then, in this form of the disease, is by no means an unfavourable sign. At the onset, as I have already said, the physical signs are the same as those of croupous pneumonia, but the course which the disease follows soon indicates its nature; when there is any doubt the sputum should be examined for tubercle bacilli.

Sub-acute pulmonary tuberculosis—chronic pulmonary tuberculisation. This is a name given to a form of phthisis which is less often met with than any of the preceding. The changes in the lungs are characterised by a drier and more gradual necrosis; there is great thickening of the alveolar walls and consequently much induration of the pulmonary tissue, in which individual granules of tubercle may or may not be distinguished by the naked eye. This form of local tuberculisation spreads through the lung from apex to base, with a well-defined grey advancing margin, immediately behind which the highly vascular but crepitant lung tissue presents a striking contrast to it. On close examination the alveolar walls are seen to be considerably thickened to some little distance, perhaps half an inch, beyond the defined margin. Further outlying patches or nodules may sometimes be observed. Dr. Powell, from whose work this description is taken, compares it to lupus of the cutaneous surface.

Patients the subject of this form of phthisis are usually of slender figures and good features. It is the consumption that novelists delight to describe, and is commonly known as a "decline." Such cases are comparatively rare. I saw a fairly typical one in the early part of this year in consultation with Dr. Burroughes of Lee. The patient was a gentleman, æt. 35, who had always enjoyed the best of health; he stated he had caught a cold just before last Christmas, but did not think much of it until three weeks before I saw him; it was then six weeks after the first symptoms. I found an extensive patch of consolidation of the upper lobe of the left lung, commencing just above the nipple, and extending as high as the second rib; there was dulness on percussion, shrill bronchial breathing and pectoriloquy, but no moist sounds. The temperature was 100° , and there was very little cough. I saw him again about a week afterwards, and in the

meantime he had been taking benzosol and phenacetin. The latter drug had reduced the temperature to 99·4°. The physical signs had extended upwards to the apex, but for an inch above the nipple the note was decidedly more resonant, and the breath sounds weaker and not so bronchial in character. There was still a complete absence of moist sounds. I saw him three times afterwards with intervals of a week ; the temperature continued the same and the physical signs altered very little, and the sounds continued quite dry. His appetite was good, and there was but little loss of strength. At the end of March he went down to Bournemouth. I saw him again a few days ago. The physical signs were much the same, repair was progressing, fibroid thickening was taking place, the diseased products being eliminated. He had gained considerably in weight, and his cough had entirely ceased. Although this case is typical from the point of physical signs, yet such cases rarely run such a favourable course. As a rule the disease is more progressive, the lung crumbles slowly away, the other lung is very prone to be attacked, and intestinal and laryngeal complications occur. The fatal termination is generally about two years from the onset, the end being rather rapid owing to the complications.

Chronic catarrhal tuberculosis of the lungs.—This is the form most commonly met with in our wards and out-patientrooms, and perhaps ninety-five per cent of our patients suffering from consumption would have to be placed under this category. Perhaps some member of our profession may cavil at the term "catarrhal tuberculosis," but it is a useful and apt one, as indicating the main change that is taking place in the lung. Tubercles are deposited in the pulmonary tissue and set up around them a catarrhal process,—a catarrhal pneumonia—which rapidly destroys the lung tissue, and it is in the course which this catarrhal process runs that the prognosis of each individual case depends. If the caseation proceeds rapidly the prognosis is unfavourable, as a rule ; whilst if the formation of fibroid tissue predominates, the outlook is much more hopeful. Under this head I bring three cases of the disease to your notice : incipient pulmonary tuberculosis, advanced pulmonary tuberculosis, and arrested pulmonary tuberculosis, and also a case of basic phthisis.

Fibroid tuberculosis of the lungs.—The term "fibroid phthisis" has unfortunately given rise to much confusion. It was first introduced by Sir Andrew Clark to indicate a fibroid disease of the lungs with cheesy deposits, but is now generally understood to mean a variety of pulmonary tuberculosis in which the fibroid element is the predominant character, for in all cases of chronic phthisis the morbid processes are of more or less mixed character. Post-mortem, single granulations or groups of tubercle of extensive areas are found, black or slatey-grey in colour, and of a dense fibroid consistence. The larger masses do not break down, but in their centres fibrous tissue develops and the special elements atrophy, so that it may be that separate tubercles can be distinguished with the naked eye at the margins only ; a few caseous or calcareous nodules may be present, but softening and cavity formation are sometimes absent (Dr. Kingston Fowler).

Fibroid tuberculosis occurs with a special frequency in men of good muscular development, but with flat chests. Such cases are marked by a generally apyrexial course, by recurrent haemorrhages, by their prolonged duration, and by the frequency with which the process undergoes arrest. There will be found increasing contraction and immobility of the affected side, traction of organs to that side, deadened percussion note and weakened breath sounds of more or less bronchial quality ; breathlessness, dragging pains, paroxysmal cough, occasional hectic, but general absence of fever.

Chronic fibroid tuberculosis arising from dusty occupations.—This very commonly happens, especially in those predisposed to phthisis ; an alveolitis is set up either through direct mechanical irritation, or perhaps more frequently through susceptibility arising from constant broncho-pulmonary irritation. Many of these cases prove to be cases of pulmonary tuberculosis. How far truly tubercular lesions are engrafted upon pulmonary fibrosis, or how far they may be a part of the original lesion owing to the inhalation of the dust already contaminated with tubercular organisms it would be difficult to say, and probably both the degree and period of tubercular infection varies much in different cases. Cases of dust phthisis are generally one-sided, and present all the features of fibroid tuberculosis.

Tubercle engrafted on bronchiectasis is occa-

sionally met with; the history as a rule is suggestive of the nature of the complaint; for a long period the trouble is confined to one lung, but sooner or later the opposite lung becomes involved, and the disease progresses rapidly.

There is one peculiarity about these cases that is interesting, namely the tendency to cerebral (embolic) abscess; this is to be particularly borne in mind in discussing operative procedure in these cases, which is occasionally suggested in order to drain the bronchiectatic cavity.

Diabetic phthisis.—Destructive disease of the lungs occurs as a frequent and fatal complication in diabetes. Statistics show ("Trans. Path. Soc." vol. xxxiv, 1893), that about thirty per cent. of cases of diabetes die from phthisis, more commonly in the second to the fourth year of the disease, and that in a still larger proportion, nearly one half, pulmonary lesions are present. The lung trouble takes the form of chronic catarrhal tuberculosis of the lungs, bacilli being present often in large numbers.

Cases and pathological specimens illustrative of the above were shown.

CLINICAL DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, North-West London Hospital,
June 18th, 1896.

DR. CUBITT LUCEY in the Chair.

Peripheral Neuritis.

Dr. HARRY CAMPBELL showed a man, æt. 40, by occupation a telegraph and telephone wire fitter, who, in view of his active life, might be termed a temperate man as regarded his consumption of alcohol. He had slight ataxia, loss of knee-jerks, sharp shooting pains from the knee to the ankle, and patches of anaesthesia. But he had not the Argyll-Robertson pupil nor any of the optic conditions met with in locomotor ataxia, and Dr. Campbell would like to hear what was the proportion of cases in which the Argyll-Robertson pupil was absent. The patient exhibited distinct wasting of the left calf, which was one and a half inches

less in circumference than its fellow, and well-marked delayed sensibility, particularly in regard to pain as contrasted with touch. Edema had also been present in the lower two-thirds of the leg, and the muscles were distinctly tender, but there was no jerking or spasm of the limbs.

Dr. GUTHRIE said there could be no doubt about the accuracy of Dr. Campbell's diagnosis; it was a case of neuritis rather than tabes. As a rule one condition marked peripheral neuritis in contradistinction to tabes, namely, the loss of dorsal flexion of the foot.

As to the preponderance of oculo-motor signs in tabes, he believed they varied a great deal. Tabes dorsalis generally started in the upper dorsal region of the cord, in which event the cilio-spinal centre was usually involved, which ended about the second dorsal nerve. In some cases the disease was higher in the cord, and affections of the pupil were very well marked, but not when the lesion was lower down. He concluded that in the present case the neuritis affected the posterior rather than the anterior nerves.

Dr. CAMPBELL, in reply, said he was not aware that there was ever any evidence of foot-drop or weakness of the extensor muscles.

Acute Temporal Periarteritis.

Dr. HARRY CAMPBELL brought forward a man, æt. 66, the subject of acute periarteritis in the vicinity of the temporal arteries. He said the condition was one of extreme rarity, so rare that he had never heard nor read of a case similar to it. When the patient was first seen, the temporal arteries and their branches on both sides were very much thickened, and their course was marked out by red lines; moreover, the vessels were very tender. Some few weeks before coming to the hospital the man complained of stiffness of the limbs, which seemed to pass upwards until it affected the arms; then he had great pain in the head, of a rheumatic type. When he presented himself the stiffness of the limbs had disappeared. The condition had improved considerably, but was still sufficiently well marked to make the case interesting.

Mr. BATTLE asked if there was any albuminuria.

Dr. GUTHRIE said he saw the case before Dr. Campbell did, in the out-patient department, and at once recognised it as a case of periarteritis. It

was fortunate that the inflammation did not extend any deeper, as local thrombosis was to be feared with an extension of inflammation to the interior. He believed the common cause of the affection was syphilis, but such a history could not be established in this case.

Dr. SIBLEY said the case was scarcely unique, because Mr. Jonathan Hutchinson had described one or two similar ones in his museum. In the patient before the Society there was little pulsation in the artery. It would be interesting to see if the pulse returns.

Dr. CAMPBELL, in reply to Mr. Battle, said there was no albuminuria, and no evidence of syphilis was forthcoming. He was not aware of Dr. Guthrie's diagnosis, and it was therefore interesting that both agreed. Pulsation could be felt in the vessel when last examined.

Hard Infecting Chancre of the Eyelid.

Mr. KENNETH CAMPBELL showed a young girl, *æt.* about 18, with a hard syphilitic sore on the upper eyelid. The history was that three months ago a young man accidentally poked his index finger beneath the upper eyelid. No result was noticed until about a month afterwards the upper eyelid began to swell. When a chancre appeared in such a situation as the present one, it presented features different from those associated with such a lesion on the genital organs. The sore had partially destroyed the lid, so that it was malignant, a character more frequently seen in hard than in soft syphilitic sores. In addition, the glands on both sides of the neck were very much enlarged, but there was no eruption on any part of the body. Anti-syphilitic treatment had produced benefit.

THE CHAIRMAN asked whether there was evidence of syphilis in the male.

Mr. JACKSON CLARKE said the case was very interesting as an example of hard chancre without an obvious eruption, a condition which, he believed, was more common in women than in men. He recently had a case in which he based his diagnosis chiefly on general enlargement of the glands, and on the good effect of mercurial treatment, and he would like to ask Mr. Campbell whether other glands than the cervical were enlarged in the patient before them. Very often a tubercular inoculated wound was difficult to distinguish from a syphilitic one, but in the former case the indura-

tion was less and the sore more passive, sometimes taking the form of warty patches with ulcers in their centre, such sores he had seen on butchers' hands. Farcy could also be mistaken for syphilis, but of course the history differed, farcy being generally ushered in by coryza. He quite agreed with Mr. Campbell's diagnosis.

Mr. CAMPBELL, in reply, said he had not been able to ascertain whether syphilis was present in the male, and did not think he would be able to find him. When the patient first came for treatment there was much pain, swelling, and spasm of the eyelid. He applied yellow sub-oxide of mercury ointment, which acted as a stimulant to the tissues, and at the same time kept them clean. She had also been taking two grains of grey powder every night in the form of a pill. He intended to continue mercurial treatment for eighteen months, so as not only to remove the present trouble, but if possible to keep under any further manifestations of the disease.

Diffuse General Vaccinia.

Dr. SIBLEY said he regretted that the mother of the child about whom he proposed to speak had not attended with the infant as she promised. The child, nine or ten weeks old, was vaccinated five weeks ago, prior to which it was quite healthy. There were two other perfectly healthy children. The vaccination was done from another child by the public vaccinator, and on the eighth day the mother noticed, in the neighbourhood of the lesions, one or two small papules, and a day or two following, other papules were seen on the forehead. In the course of the next few days an eruption covered the whole of the body, being densest on the limbs, and a fortnight after that the child was brought to him (Dr. Sibley). The infant then, at a glance, presented all the characters of syphilis, having a markedly coppery eruption over the greater part of the body, almost serpiginous in some places. He diagnosed the case as general vaccinia, and got Mr. Hutchinson to see the case. That gentleman agreed with Dr. Sibley's opinion, and published the case as one of an unusual character. Most of the gentlemen who saw it distinctly queried syphilis; but the eruption did not invade the corners of the mouth, nor, though very profuse on the buttocks, did it affect the anus, and there were no mucous tubercles. Last week three

or four fresh papules came out on its limbs, looking very much like smallpox, but the disease had not been communicated to other children in the house. The eruption had since commenced to fade, and as a result there were a number of crescents running more or less into one another, with obvious umbilication. The vaccination marks remained quite healthy, and good cicatrices had formed. The main interest of the case consisted in its diagnosis from syphilis.

Dr. WILBE asked whether calf lymph or human lymph had been used.

Mr. JACKSON CLARKE said the case was an interesting instance of a rare disease, and he trusted an opportunity would be afforded of seeing the case. He was familiar with a non-vesicular eruption which appeared on the ninth day after vaccination and then disappeared in three or four days, the onset having been attended by fever. He believed they were cases of abortive generalized vaccinia, although he knew that this opinion was not generally held.

The CHAIRMAN said he had seen several cases from calf lymph in which a small vesicular eruption appeared four or five days after inoculation and then died away. He had also seen instances such as Dr. Sibley described, but there was no secondary fever.

Dr. McEvoy asked whether there was any likelihood of varicella having complicated the case.

Dr. SIBLEY replied that the substance used was not direct calf lymph. He did not think chickenpox was present, because the eruption was so abundant, and the remainder of the family were all well. The papules continued to come out for quite five weeks. With regard to the abortive cases referred to by Mr. Clarke, he thought they were more a lichenous eruption which frequently occurred after vaccination, and in view of the variety of fluids, he thought it remarkable that diffuse generalised eruptions were not more common. The skin of the case he had narrated showed spots more than usual, because it was of a sebaceous character, and the head was scurfy.

Large Superficial Tumour of the Thigh.

Mr. BATTLE showed a tumour which he had removed from a girl æt. 18, at the Royal Free Hospital; also photographs of the case taken

before and after operation. The patient was admitted on December 10th last year, and stated that the tumour had been growing ever since she was a little girl, and was first noticed in the upper anterior part of the thigh, when it measured three inches by two. At the time of operation it formed a huge round fold of skin, extending from just in front of the anterior superior spine on the right side downwards to within ten inches of the knee and across the inner side of the thigh. The appearance presented closely resembled that of ordinary molluscum fibrosum, except that the condition of things was very much exaggerated; in fact he did not care to undertake the operation, but the patient complained about the inconvenience, the creeping sensations about it, and the fact that it perspired and smelt very badly. The operation involved a very long incision, and one inside the growth, then across to the inner side of the thigh, so that it was Z-shaped. He had to make another incision on the under surface, and it became necessary to get the two sections of skin together afterwards, which he managed successfully. The tumour showed considerable pigmentation, the surface was marked out in squares, and the skin appeared to be hypertrophied. A very uncommon feature was, that underneath the tumour, in the subcutaneous tissue, which did not appear to be hypertrophied to any extent, were a series of bodies which looked as if they were placed on nerves, and resembled strings of beads strung together—white, semi-transparent, and almost pearl-like in character. Under the microscope the appearance was simply that of a fibroma. That was interesting, because Dr. Payne had described the appearance of somewhat similar growths in cases of molluscum; but he (Mr. Battle) believed very few such cases had been recorded. The patient got well without any bad symptoms, except for a little difficulty in healing at a point where the structures did not come quite well together. Another point was that all round the tumour, the skin (which was apparently otherwise healthy), about an inch from the base of the tumour, looked as if it had been stained brown, and in one or two places there were small islands of affected tissue.

Mr. JACKSON CLARKE asked what was the course of development of the tumour. The growth reminded him of those congenital moles which

gradually extend chiefly in one direction, forming long streaks of pigmented growth in the skin. It would be interesting to know what length of time the tumour had taken to develop.

Mr. BATTLE replied that the patient told him she was born with a small place on the front of the thigh, and that when she first remembered it, it was about two inches long and half an inch wide.

Pyosalpinx.

Mr. BATTLE exhibited a pyosalpinx which he had removed from a patient, æt. 39, at the Royal Free Hospital. There was apparently a large pelvic abscess rising out of the pelvis on the left side, and extending halfway between Poupart's ligament and the umbilicus; the skin was red and oedematous, and evidently the abdominal wall was invaded by inflammatory exudation. On examination *per vaginam* the uterus was found quite fixed, and the tissues around it were very oedematous. She had the usual symptoms of abscess, the temperature rising to 101°, accompanied by general malaise, and there was a history of constipation and free fluctuation in the part. On first examining the patient there seemed to him to be no need to hurriedly open the abscess, and he thought that by keeping her quietly in bed she would improve. Her symptoms had only lasted a fortnight, and consisted of pain and constipation, but she had had no rigors or profuse sweatings. There was no history of gonorrhœa, or of a discharge from the vagina; she was a married woman, but had had no children. That was on December 16th, and he decided that if it were not better four days later he would incise the abscess. When that time had expired the patient had been brought down into the theatre, but examination under an anaesthetic showed that the tumour had diminished in size; fluctuation was very easily obtained, but the signs of inflammation and the temperature were less, and the patient was more comfortable. Examination *per vaginam* showed that there was less exudation, and a certain amount of movement of the uterus upwards could be produced. He thought it might be a pyosalpinx, and that it would be a bad course to open it then if it could first be so reduced as to admit of its removal subsequently by abdominal section. Therefore he postponed the operation, and treated the patient with hot fomentations, regulated her diet, and kept her quiet. On

February 4th, as the condition had quieted down very much, he operated, and found a tumour much larger than it looked in spirits. On opening the cavity he found recent adhesions between the tumour and the parietal peritoneum; these were easily broken down by the finger, and the tumour itself appeared as a rounded swelling, chiefly on the left side, and towards the middle line, running over the top of it, and curling round from left to right, was a hugely dilated Fallopian tube. With considerable difficulty he separated the sac from the surrounding parts, the separation from the uterus being the most troublesome. The material was most offensive greenish-yellow pus. After the removal of the tumour the peritoneal cavity was washed out with boracic lotion and a drainage-tube inserted. The patient got well without any bad symptoms, and the temperature was not even raised. Later, through some unexplained cause, the patient had an attack of cystitis, probably the result of the use of catheters. He believed that by using his discretion and postponing operation for a time he saved the patient from a serious illness, because a frequent result of premature operation on pyosalpinx was the formation of a chronic fistula. He did not know of a case in which the same course of procedure had been adopted.

THE CHAIRMAN expressed the thanks of the Society to Mr. Battle for bringing the case forward, and congratulated him on the result.

Dr. McEVoy said he would be glad if Mr. Battle would state what induced him to wait before operating, having in mind the liability of the tumour to rupture.

Mr. JACKSON CLARKE said it was often difficult to explain exactly what led to certain decisions of operators; probably it was often intuition, based on experience. Some cases of appendicitis might be compared with the present case in that respect.

Mr. BATTLE briefly replied that he could not fully state his reason for deferring the operation, but the inflammation was not so acute as it would have been had the abscess been pointing rapidly; moreover, her temperature was not so high as it would be in acute abscess, and the swelling seemed to have a very definite sac wall.

ALCOHOLISM, WITH SUGGESTIONS AS TO TREATMENT.

In the "Quarterly Journal of Inebriety," Dunham asserts that the medical treatment of alcoholism, now continued for about five days, on the average, occupies about one-tenth the time required to materially benefit these cases. The indications to be met generally are, largely, a disturbance of the nervous system, which manifests itself in an irritable disposition, sleeplessness, fits of depression, and, later, excitability, with all the phenomena of *mania-a-potu*. The digestive processes are sluggish and weak in character, the excretions are deficient, and there is a general loss of muscular tone.

The excitable stages are best controlled by chloral and bromides; or, when there is much delirium with a strong pulse, hypodermic injections of hydrobromate of hyoscine, $\frac{1}{100}$ to $\frac{1}{50}$ grain, to be repeated in six hours. In other cases smaller doses than the above will prevent the unpleasant effects of hyoscine, and yet have a good result on the cerebral congestion. Stimulants should be rapidly withdrawn, and when given should be administered in milk and other foods, but never should be given clear or "straight." If there is much nausea, it can be controlled by small doses of calomel and bismuth, frequently repeated, with nourishment, given a little at a time and often. As soon as the patient is able to take nourishment, it should be fluid in character and large in quantity, highly seasoned. If the bowels are constipated, they should be opened by injections of water and glycerin when the patient is not able to take alkaline aperients through the stomach in the ordinary manner. An infusion of digitalis, tablespoonful doses, with ten grains of citrate of potash, every four hours, will increase the urinary excretions when they are diminished. Hypodermics of the nitrate or sulphate of strychnine, with a little digitalin, are the best to overcome heart-weakness, and often relieve the delirium. Tablets made as follows for hypodermic use will be found very serviceable :

Gold and sodium chloride	...	$\frac{1}{4}$ gr.
Strychnine nitrate	...	$\frac{1}{50}$ gr.
Nitro-glycerin	...	$\frac{1}{500}$ gr.
Atropine sulph.	...	$\frac{1}{100}$ gr.
Digitalin	...	$\frac{1}{50}$ gr.
Sodium chloride	...	$\frac{1}{8}$ gr.

Cold to the head overcomes delirium when due to congestion or active hyperæmia of the meninges.

When the acute symptoms have subsided, a three or four weeks' course of curative treatment should begin. A tonic, consisting of *nux vomica*,

hydrastis, capsicum, and an infusion of gentian should be given four times a day and in full doses. Also hypodermics of the chloride of gold in solution—one-tenth of a grain to ten minims of distilled water—should be given three or four times a day.

Tablets of the above formula are good for the first week or ten days, followed by the gold solution. The platinum needle will not corrode from the gold solution, and should be used for this reason. The infusion of gentian is used because it contains less alcohol than the tincture.

Because the chloride of gold meets so many indications in the treatment of dipsomania, there is no more reason to call it a "gold cure for dipsomania" than there is to call it a "gold cure" for consumption, where it has been used with a certain degree of success; or a "gold cure" for rheumatism with deformed joints, where it has been found valuable; or a "gold cure" for paralysis of the insane, where it is one of the most efficacious remedies.

The chloride of gold the author considers one of the chief therapeutic agents in the treatment of chronic alcoholism. It has been used in cases of melancholia, hysteria, chorea, and especially nervous troubles due to syphilis. In its physiological action it seems to be a tonic for the brain and spinal cord—an alterative like mercury; it stimulates nutrition and digestion, increases secretions and excretions, and is an aphrodisiac. Its action in this respect is like that of strychnine and phosphorus.

The belief that the impotency for a time following the treatment is due to the chloride of gold, is a delusion on the part of the patient.

Strychnine tones up the nerve-centres, and the walls of the arteries are able to contract to the normal calibre, while muscular fibres recover their healthful responsiveness.

If the alcoholic should require both punishment and medicine, as they usually do, the hypodermic method, four times a day, meets the indications beautifully. If the arms swell from the effects of the hypodermics, Goulard's extract will overcome the difficulty.

In some cases, craving for drink can only be removed by treating physical ailments. Indigestion is to be treated, neuralgia and nervous exhaustion to be remedied, irregular and weak heart action to be overcome, environment and habits to be changed, syphilis and kidney trouble to receive attention. Irregular hours at meals and for sleep, the futile attempt to drink moderately, old associations in drinking, the intention to drink only beer and cider, all predispose to alcoholic excess in those who have already formed the habit.

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A CLINICAL LECTURE

ON

TETANUS.

Delivered at University Hospital, June 23, 1895,
By CHRISTOPHER HEATH, P.R.C.S.,
Holme Professor of Clinical Surgery.

GENTLEMEN,—You have just seen in the private ward the case I am now about to lecture on. The history is shortly this: a young man of 28 was admitted ten days ago, on the 13th of June, who ten days before, that is the 3rd of June, was troubled by a nail in his boot giving him some pain. On examining the ball of his foot he found a small sore, which festered; he bathed it in hot water and applied some ointment. This wound is now quite healed. On June 10th, at dinner (seven days after being injured) the patient found that he had some difficulty in swallowing, but he persisted in forcing some food down. This difficulty got worse, and on Thursday the patient found that he had pain in his chest and back, and on Friday, June 12th, he had to give up work, and some difficulty in talking became noticeable.

This patient was admitted on Saturday afternoon, June 13th, and I saw him a couple of hours after his admission. He was lying on his back, and there was marked rigidity of his abdomen, and also some rigidity of the masseter muscles. When he tried to force himself to swallow some food, a condition of spasm followed. I came to the conclusion that he was suffering from a mild attack of tetanus due to the wound in the foot, and the case, therefore, was to be classed as one of chronic tetanus. Now, tetanus is not common in this country; we have not had a case for a considerable time. I have seen one or two in private, but for some years none have been in my ward. Tetanus varies very much as to whether it is acute or chronic, traumatic or idiopathic. A case of acute traumatic tetanus is almost invariably fatal, that is the general experience; but if you get a case of

chronic tetanus, whether traumatic or idiopathic, then you may hope that the patient may convalesce and recover; that is what has happened to this patient.

What is the definition of the word tetanus? I need hardly remind you that it is derived from the Greek *τείνω* "to stretch." The best definition probably is "a powerful and painful spasm of the voluntary muscles which is long continued and uncontrollable," that is, the spasm is continuous, or, as we should call it, "tonic," as distinguished from "clonic." The spasm of tetanus is therefore continuous, there are no distinct remissions. We have of course in convulsions distinct remissions of spasm, and these we call clonic. And even in tetanus we get from time to time, in a bad case, an access of spasm of a clonic description, by which I mean that there is a sudden spasm which throws the patient perhaps on the head and heels, lasting for a minute; it then passes off, and in that way it is clonic, but the general rigidity never passes away. These convulsions are very serious, because it is in one of these attacks that the patient usually dies. You find that the patient is thrown into opisthotonus, his back is arched, and he rests on his head and heels, there is spasm of all the muscles of inspiration, his chest is fixed and he ceases to breathe. If that lasts for less than a minute, the patient, though exhausted, begins to breathe again; but if it goes on for two or three minutes, the patient falls back dead, and that is the usual conclusion of these acute cases. The patient dies from apnoea because he is unable to use his respiratory muscles. The greater number of these cases are traumatic, and the form of trauma which more frequently leads to tetanus than any other is a lacerated wound, and every now and then in a wound you may find a branch of nerve exposed. If you can cut that away so much the better for the patient, but it is not to be made out very often, and all you know is that the patient has sustained a lacerated wound, and that earth or dirt has been ground more or less into it. The meaning of that will be more clear when I speak of the bacillus.

I will quote a case of my own. A year or two ago,

the son of a medical man, out riding, was thrown from his horse and sustained a lacerated wound of the skin over the knee ; it did not involve the knee-joint, but much dirt was ground into the wound. So little was thought of the injury that the patient went up for an examination, and then he went home to his father. At his father's he fell ill and I was telegraphed for. I found that he was in violent spasms, and he died in a few hours.

The spasm of tetanus does not always take the form of opisthotonus ; there are other varieties, but the opisthotonus form is the commonest. Then there is the reverse form, when the body is bent forwards, resting on the forehead and feet, called emprosthotonus ; and another form, in which the body is bent to the side, called pleurosthotonus, but this is a very rare condition. In our patient and in all patients suffering from tetanus there is always trismus, that is, more or less spasm of the messeter muscles closing the mouth, and that was rather marked in this patient when he first came in, for there was marked rigidity of his masseters ; he has now only a little tendency to the sardonic grin—the risus sardonicus—which depends on the muscles going to the angle of the mouth, which draw it up and give it that peculiar appearance. In a mild case like this, where we have not much to go on, I trusted mainly to the rigidity of the abdominal muscles for my diagnosis. The recti abdominis appear to be the muscles which are very early affected with this spastic contraction, and they remain in that condition till the patient either recovers or dies. You have all had an opportunity of feeling this patient's abdomen, and you will have noticed that there is still rigidity present. With the rigidity there is apt to be retention of urine, for emptying of the bladder depends to a certain extent on the action of the abdominal muscles, and, therefore, in cases of acute tetanus, the bladder should always be emptied from time to time. We come finally to the spasm of the diaphragm and intercostal muscles, which generally prove fatal in these cases. Spasm of the heart does not occur in tetanus.

There are three conditions which it is convenient to contrast one with the other : Tetanus, hydrophobia, and the results of the administration of strychnia. In tetanus there is no intermission, there is no thirst, and the mind is clear. In hydrophobia there are very distinct intermissions,

there is an aversion to water, and there is marked thirst ; but in these unfortunate people with hydrophobia, when they attempt to drink, such a spasm of the pharynx is produced that they are nearly suffocated, and hence these patients refuse water. In hydrophobia there is delirium, and there are convulsions towards the end, the patient dying in a maniacal condition, in convulsions, and very often with opisthotonus. In strychnine poisoning there is no trismus ; you have spasms of all the voluntary muscles, but no marked trismus ; the cases are more rapid ; within half an hour of the administration of the dose they develop symptoms, and if called in your first question would probably be, “What has the patient recently swallowed ?” The most common method of taking it is by swallowing rat poison. For a case of suicide the patient has been lately buying most probably small portions of rat poison, and has evidently taken more or less of it. If it is a case of poisoning by someone else, then you must be on your guard as to what the patient has been given. In the well-known Rugeley poisoning case it was proved that whenever the unfortunate victim took any food which Palmer had prepared for him, in half an hour he invariably got a fresh access of spasm.

A good deal of light has been of late thrown on the etiology and pathology of tetanus. Formerly we thought it was something wrong with the medulla oblongata and spinal cord, for all that was known was that, after death from convulsions, it was generally found that there was more or less injection of the medulla oblongata and spinal cord, and of the membranes of the brain ; that was common to hydrophobia and also to strychnine poisoning and to any case of convulsions.

Ten years ago the first suggestion was made that there was a distinctive bacillus which would be found to be the cause of tetanus. A Japanese student, Kitasato, working in a German laboratory in 1889, was the first to isolate it, and show that there was such a distinctive bacillus. Now that bacillus, if you have ever the opportunity of seeing it, is remarkable in its shape, and no mistake about it is possible ; it is called the drumstick bacillus, for it has a spherical head with a straight tail coming down from it, and the name “drumstick” bacillus represents fairly well the shape of it. This bacillus is very like a bacillus which is found in mould or earth, and is known as the

earth bacillus, and there seems to be a distinct relation between the earth and the disease tetanus, because we find that tetanus is so apt to occur in any wound which has earth or dirt rubbed into it. The case I mentioned just now of the surgeon's son, who fell from his horse and had a considerable quantity of earth rubbed into the wound, is only one of many examples ; and although we have no evidence of earth bacillus in this case which I am considering now, still the man's boot would have contained dirt, and his feet probably were not very clean, therefore the inoculation might have been through the wound in the great toe. This gives a clue to the origin of tetanus and makes surgeons careful to clean every wound, because you never can tell which wound might be infected by the bacillus, which seems to be fairly common.

Up to quite lately the treatment of tetanus was most unsatisfactory. You will find that in the first place it was recommended that amputation should be performed, and we still sometimes venture to amputate. In the case of a smashed thumb sent to me some years back, tetanic spasms had begun, and I amputated the thumb to get a clean wound. I think a thumb or finger or a toe may be fairly amputated in such a case as that, but for my part I do not advise the amputation of a whole limb, because the results are usually so unsatisfactory ; in the case I mentioned of the smashed thumb the patient died. I do not think for instance that one is justified in amputating the thigh when the spasms are well marked, because the effects of an operation and the loss of blood would do more harm than good. Then besides cleaning out the wound and using every antiseptic precaution you can, you must have recourse to some drug internally. The older surgeons believed mostly in opium, and their patients often died in an insensible condition from over doses of opium. All the sedatives have been tried from time to time, and without any very good results. Then the bromides came into practice, and later, chloral ; these two drugs in combination became the fashionable treatment for this class of case, and I think altogether they are the most satisfactory. As I was not able to obtain any of the tetanus antitoxin for this case we are considering, two drops of croton oil were administered to clear the bowels, and the patient had 15 grs. of bromide and 20 grs. of chloral

every four hours. In a couple of days he was well under the influence of the drug, and he has been taking it for the rest of the week. I have now directed that to be stopped, and I have ordered him an ounce of camphor mixture ; this is only to ease the patient's mind, because he will probably like to think he is taking something. This is only to be taken till the effects of the bromide and chloral pass off, I think he then will have got well, for it is one of the chronic cases which usually do get well, but I thought it right to use the drugs, and I have not any doubt that they have done him good.

In acute tetanus there was a drug that was once much in vogue, viz., calabar bean, which is used by ophthalmic surgeons to contract the pupil. It was thought that we might get some benefit by giving it in tetanus, but I have tried it and find this, that unless you give almost poisonous doses you produce no effect at all, and when you push the drug so as to contract the pupils strongly and to produce almost poisonous effects, the patients die all the same if it is an acute case, and they get well if it is a chronic one. You will remember that some few years ago nerve-stretching was in vogue, and was going to cure all sorts of things—locomotor ataxy and many other diseases. When it was in fashion several surgeons with cases of tetanus tried stretching the nerves, and I have notes of one or two of the cases of that kind, and the inference I draw is that in a case of nerve-stretching in which the patient was suffering from acute tetanus the patient died eventually. A case that recovered was probably a chronic one, and in that case I particularly note that the stretching was not very thoroughly carried out.

The tetanus antitoxin is difficult to procure. When I heard of this case I went at once to the particular chemists who are supposed to supply it, but they were out of it ; then I communicated with Dr. Sidney Martin, and also with Mr. Horsely, who could not help me in getting the drug, but both these gentlemen came and agreed in the diagnosis, and both concurred in giving the chloral and bromide. Tizzoni and Catani are the pathologists who have worked principally at this subject, and Roux and Vallard have also studied the matter. By inoculating animals, horses and so on, they were eventually able to get immunity, and the serum of

these animals becomes the "tetanus antitoxin" and it is this that is injected. It was of no use to inject such a small quantity as Dr. Martin happened to have by him, for he told me that we should require a very large quantity of antitoxin, and therefore, not having the amount necessary for the treatment, it was not begun. Clarke, of Leicester, seems to have been the first doctor in this country to carry out the treatment, and his case recovered. He injected 100 grains in twenty-four hours, and went on for some days. Dr. Kanthack, of St. Bartholomew's Hospital, gained the other day the Jacksonian prize on the subject of tetanus : he has been working at the subject for some time, and I hope that his essay, which will shortly be published, will be widely read. But at present we know very little of this antitoxin. The cases in which it has been used are few, and the difficulty of getting the material prevents the employment of it in most cases that occur.

P.S.—The suspension of the administration of chloral and bromide was followed in twenty hours by a distinct attack of spasm with opisthotonus, while the patient was taking food. The administration was at once resumed and continued for ten days, when the drugs were reduced to half the dose, and under this the patient slowly convalesced.

A POST-GRADUATE LECTURE ON SYPHILIS AS IT AFFECTS THE LARYNX.

Delivered at the London Throat Hospital, Great Portland Street, March 20, 1896, by

W. MACNEILL WHISTLER, M.D., M.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—It is not my purpose in a lecture of this kind to go much into the earlier investigations which were made in connection with syphilis in the larynx. Suffice it to say that, contrasted with those that had been made in regard to the manifestations of syphilis in other organs, the question as to how the larynx was affected in the evolution of this disease was, for long years,

practically a matter of inference. In the days before the laryngoscope was introduced, some of the appearances in the later stages of syphilis as seen in post-mortem examinations, are described, including swellings, tumours, and deep ulcers. The morbid changes during the active progress of the disease, however, could be but a matter of surmise then. So we find, looking back to the literature of the subject such statements as the following :—

"It is very plain that when the syphilitic ulceration is confined to the larynx, and notably to the ventricles, it cannot be distinguished by any special sign, and one can only recognise it to be syphilitic by means of the antecedents of the patient, and by the symptoms which may at the same time exist upon the skin, in the bones, and so forth."

Again : "*Syphilitic laryngitis, in the great majority of cases, is the extension of the lesions of the pharynx and nasal fossæ, which are so common in syphilis. It is important, therefore, to take into consideration this special march of syphilitic laryngitis, for experience shows that ordinarily the larynx is the seat of lesions which are analogous to those which are previously noted in the throat. For instance, a non-ulcerating laryngitis follows upon an erythematous syphilitide of the nasal fossæ and of the pharynx ; while, on the other hand, one has reason to presume that there exists in the larynx syphilitic ulceration and necrosis when one sees an analogous lesion in the nasal fossæ, and that the tonsils and velum palati have been deeply ulcerated."*"

These were the views published by Rousseau and Belloc now sixty years ago, in their most interesting treatise on the subject, entitled *Syphilitic Laryngeal Phthisis*. It is only since the introduction of the laryngoscope that any accurate or reliable information has been gained on this subject. Even after this, and in the earlier years of the laryngoscope, opinions differed a good deal, especially as to the frequency and character of the laryngeal signs of syphilis in the secondary period.

I will not encroach upon our time to-night by entering into all these details, interesting as they are ; I will merely state that on the one hand it was asserted that the surface appearances in the larynx corresponded to those on the skin, both in their type and also as being directly associated eruptions.

On the other hand, this view that definite lesions were found in the larynx corresponding to the

divisions of syphilis into secondary and tertiary periods was opposed. So it was proposed to divide syphilitic laryngitis into two forms only, namely, the non-ulcerated and the ulcerated, either of which might appear at any period of the disease: the former including hyperæmia, œdema, and hypertrophy; the latter comprising the various forms of ulcers, together with perichondritis, caries, and necrosis. It is not surprising that in the pre-laryngoscopic stage, with lack of means to see what really took place in this organ, it should thus have been argued as most probable that the surface evidences of syphilis in the eruptive stage might be accepted as identical with those on the skin and mucous membranes in other parts. It is also not unreasonable to infer that, in the earlier efforts to test the truth of these arguments, the first investigators should themselves have been somewhat prejudiced by these very inferences, and that they should have dwelt upon minutest differences of detail to prove or disprove those assertions. The interesting and important fact which we elicit from all this is, that while they differed in detail, their united studies established the fact that the main characteristics of syphilis were to be found in the larynx as elsewhere. Unquestionably, in the course of syphilis as it affects the larynx, the earlier changes may not be strictly compared as absolutely identical with the eruptions on cutaneous surfaces as some sought to do, but this is true also of the buccal and pharyngeal regions. We do not find a typical roseola of the mouth or throat corresponding with this form of syphilis on the skin. Neither is a squamous eruption of the surface necessarily or even usually accompanied by syphilitic psoriasis of the tongue. There can be no doubt, though, that certain phenomena of a more superficial nature do occur in the larynx in the earlier periods of the disease, while deeper and more destructive ones mark a more advanced stage, and this with sufficient constancy to justify certain types being grouped together to represent certain periods. Nor can this be overthrown by the negative evidence of the presence in individual cases of comparatively trivial congestions of the larynx associated with advanced tertiary manifestations in other parts.

It is not to be denied that, as cases present themselves to us in our practice, and especially in the more advanced stages, they may frequently represent, as more salient features, ulcerative pro-

cesses, or the reverse; but, unless it be for the simplicity of such a broad classification of laryngeal syphilis as I have referred to, there appears to be no more real reason for subdividing these specific lesions of the larynx into non-ulcerating and ulcerating than there would be for the same classification as regards the more general lesions. The usual division of syphilitic affections of the larynx into secondary and tertiary only is, in my opinion, not an altogether satisfactory classification either. When, many years ago, I was making special investigations into the subject of laryngeal syphilis, I noted an important class of cases in which the laryngoscopic appearances, as well as the course they ran, differentiated them from the earlier and the later forms; and when I published the results of my examinations in 1879, I described this type under the name "relapsing ulcerative laryngitis of the *intermediate* period" (speaking of these changes from a clinical point of view). This subdivision which I made was a good deal commented on at that time, and while there were those who admitted that it accurately defined the condition which in practice they had constantly met with, others advanced as an argument against the subdivision that the lesions I had described as characteristic of the intermediate stage might well, on the one hand, be considered secondary, and on the other hand might merge very closely into what was admittedly the tertiary stage. I do not think I could explain to you more clearly my reason for adopting this intermediate subdivision than by quoting to you that objection which was made against it at the time.

It is just because the laryngeal lesions in this group of cases are associated with, and mark, so to say, a borderland between the conditions which are found in both the earlier and the later stages, termed secondary and tertiary, while they present characters distinct from either, that I described them as marking a period of transition. And I still feel that this classification expresses more accurately and simply what the laryngoscope reveals to us of the gradations which occur during the progress of syphilis as it is seen in the larynx. These may be grouped together as initial inflammation of a superficial nature, which may end in complete resolution, leaving no trace of its former existence, but which, if not so arrested, is followed sooner or later by graver tissue changes,

marked by varying degrees of ulceration, and necrotic processes associated also with proliferation of tissue, leading in the end to serious structural deformity. These different conditions I will now describe to you very briefly as I have met with them ; dividing them into the following classes :—

(a) Earliest manifestations belonging to the eruptive period, associated essentially with the general secondary symptoms, viz. catarrhal congestions and mucous patches.

(b) More chronic inflammation, occupying, as it were, the period of transition, the signs of which are diffuse redness, thickening, and ragged ulceration, especially of the vocal cords—*relapsing ulcerative laryngitis* of the intermediate period.

(c) Later manifestations, either co-existing or not with general tertiary symptoms, but not associated with those belonging to the secondary period :—(1) Acute gummatous inflammation. (2) Relapsing laryngitis of the tertiary period. (3) Chronic fibroid.

Now as regards those congestions of the larynx which occur in early syphilis, and which simulate ordinary catarrhs, I need not say much. They are essentially superficial in character, like those in the nose and pharynx. Increased redness, which varies in intensity and tone, is their chief feature. When unequally distributed, it may give to the membrane a mottled appearance, the epiglottis is redder than usual, its mucous membrane is no longer so transparent, the yellowish hue of the epiglottis is lost, while tortuous vessels are seen running over its surface. In some cases the vocal cords are chiefly affected ; sometimes they are pinkish and dingy-looking, at others deep red and dry. They may be merely streaked with a few red lines, or the mucous membrane is occasionally eroded, and with this there is a lack of proper tension, with defective approximation, and this leads to alteration of voice. All that these patients suffer from, as a rule, is hoarseness.

Occasionally one meets with intense redness in the interior of the larynx, with swelling of the ventricular bands and arytenoids ; accompanied by pain and cough. When such complications arise, they are generally due at this stage to some super-added cause other than the syphilitic poison, such as either cold, excess in drinking or smoking, or the too constant exercise of the voice. One meets with a good many such cases, which resemble acute

laryngeal catarrh, and are relieved by the same measures. Confinement to a warm room and the inhalation of benzoin or conium at intervals of two or three hours will relieve any such acute symptoms more quickly than mercury would do *without those aids*. They should be, therefore, always added to the mercurial treatment in all such cases, for it is more than ever important that a laryngeal inflammation be reduced as quickly as possible when it occurs in the course of syphilis.

I come now to another type of syphilitic laryngeal congestion of more special character, which occurs at precisely the same period as the one I have just referred to, that of general eruption,—as a rule, during the first two years. The redness is equally diffused over the laryngeal surfaces like an erysipelatous flush or erythema. No small vessels are seen running over its surface. Together with this there is swelling, or puffiness perhaps better describes the condition, as though there were a certain amount of œdema or infiltration associated with it. This is most striking on the epiglottis ; its edges are rounded and cord-like instead of being thin and sharply defined. The epiglottis has a peculiar fleshy look, as contrasted with the delicate tint of the normal epiglottis. Sometimes this redness extends over the whole mucous surface of the larynx. When, on the other hand, it is limited to certain parts—and this is the case sometimes—the line of demarcation is well defined. The colour is at first rosy and somewhat opalescent-looking, gradually shading into a deeper red. In certain cases it is more distinctly patchy or spotted, like the condition alluded to as a laryngeal roseola. In some cases of this kind, in which I found this more strikingly defined, the laryngeal affection was associated with a squamous syphilide, and not with a roseolous eruption. From none of these laryngeal signs or symptoms, however suspicious they may be, could one absolutely pronounce the case syphilitic if they are the only ones we have to go by, and one must look for other associated constitutional manifestations to confirm the diagnosis. If you will do this, you will find in a syphilitic patient either enlarged glands, inguinal and cervical, associated with a roseolous, papular, or squamous eruption on the skin, or mucous patches. In all cases of syphilis of the larynx at this period other general lesions will not be

wanting. It will be exceptional if we do not find some of them at the first examination; and we will be sure to do so if the patient remains under observation for a few weeks. Among these, those which are most constant are mucous patches of the mouth and pharynx. Taking these last-named phenomena as typical of this period, we come now to the question, do mucous patches or "condylomata" of some authors occur in the larynx; and if they do, are they at all in proportion to those appearing elsewhere? These points gave rise to much argument at one time, in which some eminent authorities strongly contested that the full proof of the existence of laryngeal condylomata was not established, while others denied their development in the larynx altogether. The published reports, however, of the greater number of observers, by now, bear proof of their presence in this organ. Yet, while nearly all laryngoscopists testify to having seen them, the general consensus of opinion is that these patches, so common on other mucous surfaces, are of rarer occurrence in the larynx. My own observation, made at the date I have mentioned, and carried out for several consecutive years, convinced me that this essentially secondary manifestation has its place in the larynx as elsewhere. More than that, I am sure that although these mucous patches are rare when compared with the numberless ones in the mouth and pharynx, still they do occur more often than has been allowed by some authorities. They vary in their appearance according to their situations, and in this they resemble other mucous patches; though differing from each other in certain respects they have one type in common, namely, that in their primary state they are all more or less papular. This would serve to distinguish them from superficial ulcers or erosions associated with ordinary catarrh. When they ulcerate, the ulcers are more regular in outline; they are more distinctly circular and circumscribed than are the erosions sometimes associated with ordinary catarrhal laryngitis. Take, for instance, an inflammation of the larynx of non-specific nature, the patient complaining of hoarseness, dryness of the throat, and a constant desire to clear it—not uncommon symptoms in such cases. If you examine such a larynx, you will probably find, together with general congestion, more or less thickening of the vocal cords, which will be bright

red and raw-looking, with very likely a certain amount of tenacious adherent mucus. The patches of mucus might mislead you, but only for a short time, for they could be brushed off or coughed up, leaving the cord as I have described. The ulcerated mucous patch will either be a papule with an ulcerated summit, or an oval grey ulcer which may be somewhat ragged at the edges. It never attains any depth, and as a rule yields readily enough to treatment. Moreover, comparatively recent non-specific inflammation of the larynx does not usually tend to ulceration; in fact, so far as my observations go, I do not hesitate to say that generally, when true and persistent ulceration is met with in the larynx, time will usually show that this is due to some specific cause other than simple inflammation in that class of cases called "chronic laryngitis." When such ulceration is met with in these chronic doubtful cases, it might be confounded with some of the later syphilitic ulcers, but not that with an ulcerating mucous patch. Though the symptoms accompanying these early indications of laryngeal syphilis are often comparatively trivial, they may, if unrecognised and neglected, be the forerunners of grave disturbance, leading not only to permanent impairment of voice, but also to painful and dangerous interference with respiration.

The affection of the larynx which I have next to describe to you is one of more chronic inflammation, in which the signs are diffuse redness, thickening, and ragged ulceration, especially of the vocal cords. I show you some drawings which were taken from cases of my own. This occupies an



intermediate line between the early and later lesions of syphilis of the larynx. It may occur close in the wake of the former, and be the immediate outcome of the catarrhs and mucous patches of the larynx which I have described; or it may show itself at times three or four years after the primary sore, and even, though exceptionally, later still. In the first instance there would be still remaining, as an accompanying manifestation, some more

or less general eruption on the skin, with mucous patches of the mouth ; while in the other extreme there may be syphilitic tubercles limited to the arms or legs, periosteal inflammation, or scars from ulcerating syphilides, with ulcers of the fauces and chronic glossitis. This condition of the larynx is developed in somewhat the following way.

A patient will have a laryngeal catarrh in the first few months of the disease ; mucous patches of the cords appear, and recurring often, take on ulceration. Under treatment these ulcers heal more or less perfectly : several relapses occur, the cords become now more deeply reddened, they lose their flat appearance, and get rounded and roughened on their surfaces as though worm-eaten. The ulcers again break down, their edges swell and thicken, and are bordered at times by vegetations. The ventricular bands and interarytænoid fold participate in this inflammation ; they are thickened, their surfaces are roughened, and warty growths may now spring up and form spur-like projections over the vocal cord. This is an accurate description of the growth and development of this affection at an early stage. Again, the case may be this : a woman, married ten years before, has sores on the vulva and rashes on the body. The first child, born fifteen months after marriage, has rashes on its body, and dies at three months. The second one, born a year after this, dwindles away and dies in a few weeks. The mother next has a bad sore throat between three and four years after the primary lesion, and subsequently has two miscarriages. All this time she is ailing, but she has no return of eruptions, and her throat is only occasionally sore. Nothing more occurs until another four years have passed ; then she has headaches, and her throat becomes bad again. She is treated for it and gets better, but never quite well, and then her breathing becomes embarrassed.

This is not an imaginary case ; I have only been relating to you the history of a woman who came to me for treatment some years ago, with thickened and ulcerated cords, like those I have just described to you as following on ulcerating mucous patches. The condition of the larynx in the former dated eight months after infection, while in this woman's case ten years had elapsed since infection when I first saw her, though the

laryngeal symptoms she suffered from were of long standing. She presented herself for treatment for hoarseness and dyspnoea, and told me her story as I have told it to you. She had superficial ulceration of the tongue, deep redness of the larynx, with redness and picked-out ulcers of the vocal cords, and thickening of the interarytænoid fold. She was put upon iodide of potassium, but this did not give quick relief, and I soon was obliged to take her into the hospital, as I thought she would require tracheotomy. There was inflammation and swelling of the ventricular band and interarytænoid fold. The vocal cords were highly inflamed, deep red, and ulcerated. They were thickened and rigid, and their movements in abduction were impaired, so that the chink of the glottis was not more than half the normal size in inspiration. There was no acute œdema. Beside the dyspnoea she was almost aphonic, and had a bad cough and pain in the larynx. She recovered rapidly under mercury and iodide of potassium, and was discharged cured in a month. She attended subsequently for relapses, which occurred at intervals for two years. Then she was discharged cured.

Now if you have followed me through these descriptions, you will have seen that they are all identical in their main features. They differ from the class of cases which I first referred to : the ulcers are deeper, ragged, and with thickened edges ; they are small and irregular in outline, often multiple, and the vocal cords upon which they are situated look as if pieces had been torn out of them. Still, they are comparatively superficial, and are not usually accompanied by perichondritis, nor by necrosis of the cartilages as are the burrowing ulcers of the later period. There is thickening of tissue, and this infiltration is not only diffuse leading to hypertrophy of the ventricular bands, vocal cords, and other parts of the larynx, but it is also associated sometimes with more circumscribed outgrowth in the form of warts and nodules. These latter do not differ in appearance from those arising from other causes. There is nothing specially indicative of syphilis in their presence. They are found in laryngeal phthisis, though may be not so frequently, as there the destructive process is more rapid. Some authorities have considered the presence of warty growths, in phthisical ulcers of the larynx, to point to a possible association of syphilis in such cases. One meets with

them, however, not infrequently under such circumstances where there is neither a history of syphilis nor any manifestation of such a taint. Moreover, they are met with in ordinary chronic inflammation of the larynx. Owing to these more serious changes in the larynx, the functions of this organ are more impaired, and, as might be expected, the symptoms from which the patients suffer are graver. The hoarseness is often extreme, not infrequently with some difficulty of breathing, with troublesome cough and pain in the larynx. Every now and then acute attacks occur, accompanied by intense swelling of the tissue, producing great difficulty in breathing; but this is exceptional, and even the most threatening cases frequently escape tracheotomy. Besides the greater degree of chronicity of this affection, relapses are more common, and these relapses are sometimes similar in every way to the preceding attacks. In this way you may meet with cases of this relapsing ulcerative laryngitis of the intermediate period extending over several months, sometimes over a year or two, with slow changes in the larynx. If the affection be arrested in time the ulcers of the cord cicatrize, leaving a fairly smooth edge, the thickening subsides, the swelling passes off, and the voice is restored. But if the disease be allowed to progress, large portions of the cord are lost, their movements are irremediably impaired, and permanent hoarseness is the result. Still further, they may be the forerunners of those characteristic lesions of the larynx belonging to the tertiary period, or period of gummatous productions.

We deal now no longer with hyperæmia—light and transient inflammation—or with those ulcerations which, however persistent, begin on the surface, and at their worst remain comparatively superficial. From now the course of syphilis as it affects the larynx is marked by more deeply-seated alterations, which have their origin in the connective-tissue elements, and are associated with the formation of a characteristic neoplasm. This may be disseminated, or more strictly circumscribed, giving rise in the first instance to diffuse and chronic hyperplasia, and to the formation of a fibroid growth, or in the other it appears under the form of nodules and tubercles, known as gummata. Under this latter condition, and when massed together in quantity,

it has a less vitality, and its elements undergo necrotic changes, resulting in ulceration.

So chronic proliferation of tissue and advancing ulceration are the types of the latest encroachments of syphilis upon the larynx. These processes may exist separately, though they are more often combined, and, according to the predominance of either, present those serious signs of progressive growth which lead to narrowing or deep destructive ulceration, which in the extreme ends in irremediable deformity. In fact, the main features of the different phases of tertiary syphilis of the larynx, and the modifications of its progress, depend greatly upon the balance which is established between these two morbid processes. For the better consideration of this question, and to make my meaning clearer, I would divide tertiary syphilitic manifestations affecting the larynx into two classes—the acute and chronic.

The pathology of syphilis as it affects the larynx is the pathology of general syphilis. Its peculiar type when it attacks the larynx is derived from the structural elements and anatomical relations of the component parts of this organ. We have here occupying an extremely limited area, a cartilaginous framework, lined with mucous membrane: a submucous cellular tissue supplemented by a fibro-elastic layer, in close relation with articular structures, whose movements regulate the action of the vocal cords in their phonetic and respiratory functions: also muscles which supply the motor mechanism; nerves—sensory and motor, together with a plentiful vascular supply. What more ample scope could be afforded for the ravages of syphilis than this close association of structure? Taken singly, we have here all the elements which by the action of the syphilitic virus, not only bring about organic changes—hyperplastic and destructive—disease of cartilage, serous infiltration, and suppuration, leading to œdema and abscess, and articular ankylosis; but when combined lead to most urgent complications, either direct and structural, or those which indirectly disturb innervation. When, in addition, we remember that the larynx is, so to say, the gateway to the lower respiratory organs, we have a full explanation of those most serious and urgent symptoms which continually complicate the course of the later stages of laryngeal syphilis.

Gummata of the larynx, as seen by the laryngo-

scope, are found as circumscribed tumefactions on the surface, either solid or ulcerating nodular eminences, varying in size, sometimes having dimensions of small shot or hemp-seed, at others forming irregular ovoid or spherical tumours of larger proportions. The smaller growths are usually multiple; and grouped together they appear as prominent nodulations, the surface hue of which is generally modified by that of the surrounding surface. When this is much congested we find these granulations deep red, or shading from this colour to a reddish-grey or yellow tone. But when they are associated with little or no general inflammation they may at times be quite pale and indolent-looking. As a rule, though, the mucous membrane covering them is abnormally red. In this granular form they are not so prone to ulcerate, but remain sometimes as small tumours, frequently persisting for a long time,—then disappearing under appropriate treatment. Again, they form the centre of more diffuse tissue proliferation. Laryngoscopically, they present the appearances which are spoken of as protuberances, roughness and irregularity of the surface, when not combined with ulceration. It is not infrequent, however, for these growths to form around the borders of more sluggish tertiary ulcers. They may occupy any part of the larynx, and I should not say, from my own observation, that they seem to have a predilection for any special portion. They are commonly found in chronic syphilis of the larynx, on the epiglottis, nodulating and distorting its outline and thickening its substance. On the ventricular bands and vocal cords they appear as swellings or nodes, following the outlines of these either in limited segments, forming sometimes polypoid vegetations, or leading to general thickening. Although these smaller gummatous formations may thus remain as more or less chronic outgrowths of solid or semi-solid consistence, they, like the more circumscribed and acute gummata, pursue, readily enough, an opposite course, and softening, lead to ulceration. When massed together they are the essential cause of destructive ulceration of the epiglottis, in the progress of which both the soft tissues and the cartilages are involved, resulting in a permanent loss of large portions, and at times even of the whole structure. In this way are brought about the even more grave ulcerative changes

of the vocal cords, which are followed by permanent loss of voice, and which in some cases lead to adhesions and web formations, which diminish dangerously the respiratory space.

On the anterior and upper surface of the interarytænoid fold, gummata are seen in the laryngoscopic image as either thick ridge-like prominences or rounded bosses on its surface, or as warty eminences, which project as outshoots over the posterior portion of the glottis. The thickening, roughness, and rigidity which ensue interfere very materially with the perfect adduction of the vocal cords, and is one cause of the rough, hoarse voice, which is so characteristic a symptom in these cases. Again, encroaching as it does on the glottic space, it seriously aggravates the difficulty of breathing during acute inflammatory attacks which so often arise and recur in these cases. This is the picture of these neoplasms in this region during their stage of growth; but here again, as on the rest of the laryngeal surface, they break down, and the ulcers which result have the typical character of those found in the pharynx, with infiltrated, swollen, and livid red borders, short-cut and undermined edges, and a foul, yellowish grey disintegrating surface. The march of these ulcers is progressive, in their progress they are destructive, and, burrowing deeply, they lead to complications by involving the surrounding structures—perichondrial inflammation with œdema and suppuration, necrosis of cartilage, which may lead to extrusion of the necrosed portion, ankylosis of the joints, destruction of muscles, and cicatricial contractions. Nowhere are the cartilages more liable to be affected by syphilis than in the larynx. Indeed, we may put it down as a rule that in syphilitic laryngitis, when it has reached the stage I am now speaking of, perichondritis never fails to intercur at some period, unless the disease should fortunately be quickly arrested by treatment. It attacks most frequently the cricoid cartilage over its posterior surface, and extends usually from there to one or both arytenoids. When very acute and extensive it may be bilateral, but supervening upon chronic laryngeal disease it is often seen to affect a more limited area, and its course is then restricted to one side. The inflammation not only leads to increased swelling, but also to suppuration beneath the perichondrium, forming a deep-seated abscess. This discharges either

into the pharynx, especially if it points chiefly over the posterior surface of the cricoid, or (infiltrating the crico-arytænoid articulation) the pus finds its way to the surface in the interior of the larynx ; or, burrowing more widely outwards along the outside of the thyroid cartilage, discharges on the anterior and lateral cervical region, where it gives rise to more or less intractable sinuses.

Associated with this we find serous exudation, and so œdema is the essential symptom, which is superadded to, and, so to say, goes side by side with the course of perichondritis, and intensifies the urgent symptoms which result from it. œdema occurs especially over the under surface of the epiglottis, over the arytenoid region, and it is very typically marked in the ary-epiglottic folds. By the laryngoscope it may be readily recognised as a semi-translucent or opalescent swelling of globular form, the colour of which is pale red, or greyish where the distension is greatest, shelving at its borders into the intense redness or lividity of the surface, which, with diffuse swelling, are the laryngoscopic appearances of perichondritis.



Both these processes may be more indolent in character and more permanent, and then the signs are chronic tumefaction—brawny red swelling—involving the arytenoid region, the ary-epiglottic folds, and the ventricular bands.

In those forms of more limited perichondritis which are associated with the acute onset of later laryngeal syphilis the inflammation may be either arrested before the suppurative stage, or, even going further, with the evacuation of the acute abscess, a cure may ensue with comparatively little and no grave result. These cases, however, mark the happy exceptions, and in a large number of cases of perichondrial inflammation with suppuration, recurrent abscesses are the rule, with continued repetition of acute distress ; and the inflammatory processes and tissue changes, extending end in

permanent hyperplasia and ankylosis : the impairment or complete disorganisation of the articulations being due, on the one hand, to adhesive inflammation, or thickening, to the extrusion of a necrosed arytenoid cartilage, or to the destructive ulceration ; together with rigidity or atrophy of the muscles governing their movements.

So far I have enumerated to you the various morbid changes which arise from syphilis in its progressive course, but even the processes of repair now are not free from gravity, and offer new elements of danger. The contractile tendency of the cicatrices of syphilitic ulcers is a well-known characteristic, and by this contraction the larynx is most gravely affected. Not only is its lumen narrowed in this way by the cicatricial contraction of the ulcers and by the membranous adhesions which may result, but the contractions of pharyngeal ulcerations which occur at this period may, in rarer cases, lead to such distortion and extreme narrowing of space that the larynx is almost hidden by these cicatricial bands, and its respiratory functions most seriously disturbed.

In the earliest stage of syphilitic laryngitis the symptoms are chiefly those of surface irritation—temporary soreness at the worst—with cough and hoarseness, which symptoms are more intense in the *relapsing ulcerative* form. In these latest stages, belonging to the tertiary period, we find persistent alteration of voice, varying from gruff hoarseness to almost complete aphonia, deep-seated pain—especially during the course of perichondritis,—dysphagia when there is much swelling combined with abscess ; but of all the symptoms the most serious one now is dyspnoea, the attacks being due to sudden and acute inflammation, or to progressive stenosis from fibroid growth, to cicatricial contraction, and to web formations.

It would be waste of time to insist to you upon the importance of constitutional treatment being associated with local measures. To get these cases quickly under the influence of mercury and iodide of potassium is the first requisite, either to prevent or to arrest the emergencies that may arise. You may use any form of mercury, varying it as may be indicated. Where a more prolonged mercurial treatment is called for in the earlier stage, small doses of grey powder—a grain and a half to two grains three times daily—is very beneficial.

The tannate of mercury is also one of the best forms—given three times a day in doses of $1\frac{1}{2}$ grains. This is not so apt to produce diarrhoea. Either of these may be combined with Dover's powder to avert this. In the lesions of the intermediate and tertiary stages, iodide of potassium combined with mercury is required—say liquor hydrargyri perchloridi $\frac{3}{4}$ j, potassi iodidi gr. x., increasing to gr. xv. or xx. in one ounce of water three times daily. In the graver and more persistent cases, mercurial inunction, associated with iodide of potassium internally, in full doses gives excellent results. In urgent cases of dyspncea, due to œdema of the larynx, scarification of the acute swelling may afford complete relief. I have known it do so in many cases; but when this and the other treatment I have spoken of fail to overcome the laryngeal obstruction, I need hardly say to you that tracheotomy should not be delayed.

For the more superficial inflammations, you will find a solution of chloride of zinc in water, fifteen or thirty grains to the ounce, a very serviceable application. Sulphate of copper, fifteen grains to the ounce of water, acts very beneficially in cases of mucous patches; so does a solution of nitrate of silver, ten grains to the ounce. A strong solution of this salt, one drachm to the ounce of water, gives the best results in *chronic* ulcerations with thickening, but it must be applied with care, and is not applicable to *acute* inflammatory conditions, owing to the laryngeal spasm it is then apt to induce. Iodoform, by insufflation, may also be applied to the ulcerated surfaces. For the purpose of applying iodoform more constantly than can as a rule be done by insufflation, I introduced several years ago its use in the form of pastilles—one to two grains of iodoform to each pastille, made up with a special gelatine basis. I show you these now; and also some containing morphia and bismuth, prepared in the same way. These latter are extremely useful to relieve pain and cough. Insufflations of morphia— $\frac{1}{8}$ or $\frac{1}{4}$ grain to $\frac{1}{2}$ grain of powdered starch—are also one of the best applications.

Chronic stenosis of the larynx of the tertiary period, resulting from cicatrices, webs and thickening—in cases amenable to operation—can only be overcome by incision, dilatation, and extirpation of growth.

This subject I must defer to a future lecture.

CLINICAL NOTES.

WITH DR. F. J. SMITH IN THE WARDS OF THE LONDON HOSPITAL.

Gastric Complication in Post-diphtheritic Paralysis.

THIS is a case of a lad who six months ago complained of a sore throat, but was only admitted to hospital the other day for a surgical opinion. The question of diagnosis hardly enters into the case; the sore throat was fairly obviously diphtheritic, and he was handed over to the medical side for investigation and treatment. The interest in the case arises from the gastric symptoms, presumably from implication of the pneumogastric nerve, so that we had at first some trouble in feeding him, but that passed off with a little care in diet, and he was doing pretty well till two days ago, when he apparently developed whooping-cough. There is a rise in his temperature, and a recrudescence of the old gastric trouble. The treatment of diphtheritic paralysis consists in good feeding with a little strychnine. The difficulty here was to get the food into the stomach and to prevent it being ejected by vomiting; slops and soft diet in very small quantities at a time were the chief helps in this direction. Since the antitoxin treatment has been adopted, cases of post-diphtheritic paralysis are more frequent. This is due to the fact that so many more of all cases of diphtheria recover now than did formerly, so that cases showing paralysis must naturally be more common in simple proportion; and we must also remember that the antitoxin cannot actually repair nerves which are already damaged, though it unquestionably prevents a fatal issue in many such cases. This early damage to nerves emphasises for us the importance of the early application of the antitoxin treatment; for if we can only apply it early enough it will neutralise the toxin, and therefore prevent nerve degeneration. In the average hospital case, several days advanced in the disease, a large amount of toxin is present, and the damage to nerves is already done, they are actually degenerated, and the antitoxin cannot undo the damage; all it can do is to prevent its getting worse. More children recover now, and

consequently there are more paralysis cases than formerly. I think this comparative frequency of post-diphtheritic paralysis is most eloquent testimony to the life-saving properties of antitoxin, and to the rapidly growing favour with which the method of treatment is being received.

Addison's Disease.

The next is a case of Addison's disease. It is a somewhat rare complaint, and the essential points in the diagnosis are an extremely feeble pulse, usually, but not invariably, associated with the typical sickness and bronzing. This case still shows the bronzing very well ; it has much deepened under observation. It is curious to note how sharply defined is the region covered by an old belladonna plaster, showing the local influence of pressure in shaping the pigmented areas. But the sickness is now not so prominent,—it had, in fact, quite ceased when he came in ; the very feeble pulse, too, has markedly improved under the treatment adopted, which has been supra-renal preparations. This is the second time he has been an in-patient under my care. At the end of his first stay in hospital (of six weeks' duration), he went out certainly much improved ; but, unfortunately, while at the seaside the east wind did him harm—it seemed to throw him back, and he returned to us very weak and depressed, but the sickness has never returned with its old violence.

[P.S.—Very shortly after the above note was written the patient died, as they usually do, in a syncopal manner. The autopsy proved the correctness of the diagnosis, as typical caseous supra-renals were found, and his death gives me the opportunity of commenting on the treatment by gland preparations. Most certainly they seemed to keep the sickness at bay, and the pulse gained remarkably in volume and strength, so that we may claim remission in symptoms and gain in comfort to the patient as results of the treatment. But, on the other hand, I feel bound to record my opinion that from a curative point of view we gained absolutely nothing (except experience), and death was not even delayed to the best of my belief. Any fuller discussion of the problem would be out of place in a note like this, and the case must be left as a contribution to fuller statistics awaiting further experience confirmatory of my opinion or opposing it.]

Diuretin and Morbus Cordis.

The next is a case of morbus cordis in which, after using digitalis with temporary but diminishing effect, I tried diuretin, a drug that has been lately offered to the profession. My conclusion has been that it has here failed in causing any definite and marked benefit. The facts are : The patient was passing 20 oz. of urine ; after taking diuretin the amount rose to 34 oz., and I thought that diuretin was going to be very successful in his case, but now the urine has gone down in spite of the diuretin being pushed. Diuretin is given in a powder, 15 grs. of which are easily dissolved in an ounce of water. The legs now do not show much swelling, but that improvement is attributed more to the use of Southeys' tubes than to the use of the diuretin. The patient died shortly afterwards, and presented a very rare condition, a small aortic aneurysm communicating with the pulmonary artery, unsuspected during life. I hope to give diuretin another chance.

Feeding in Typhoid.

The next is a case of typhoid. I do not believe in waiting for the temperature to remain down long before giving these patients food. As soon as the temperature is down I order jelly or fluid custard, the intention being well illustrated in this case, viz. to give strength to fight a possible relapse. This man had a temperature of 102° (once it was up to 105°) for five weeks and a half before admission ; for another fortnight after admission the temperature showed considerable variation between 102° and 103·5°. The diagnosis was obscure as he came from a tropical country, but balancing the evidence I concluded that it was typhoid, or malarial typhoid, or typho-malaria, according to choice of terms, with a common basis of intestinal ulceration. As soon as the temperature was normal we allowed him as much food as he seemed to wish for, and the success of my plan is happily illustrated. After an apyrexial period of eleven days he had a relapse lasting fourteen days ; still he has pulled through it without any serious damage. Now, after seven and a half weeks' serious pyrexial illness, exhaustion must be and was in this case most extreme, and without the week's feeding I do not think he could possibly have survived. Looked at from the view of ordinary digestion, I cannot myself see how a little

soft crumb of bread, as opposed to the crust, can do harm in these cases; it must get into a smooth pultaceous condition before arriving at the ulcerated patch in the intestines; similarly with pounded meat, it appears harmless. My ordinary plan is to allow blanc-mange and jellies as soon as the temperature falls. I do not wish to be misunderstood as forcing this food on typhoid patients, but I allow it if the appetite asks for it.

Cirrhosis of the Liver and Abdominal Section.

The next is a case of alcoholic cirrhosis of the liver and dropsy in an old woman. The point I wish to mention in this connection is that of the treatment of ascites from whatever cause arising: I have determined to submit all such cases to surgical treatment by incision as soon as removal of the fluid seems distinctly indicated. My reasons are, first, that the risk of the exploratory abdominal incision is less than that of blindly and somewhat violently running in a trocar and cannula; secondly, that the diagnosis, if in doubt, can be cleared up to a certainty, for three fingers in the abdominal cavity give you a chance of making a diagnosis which external abdominal palpation cannot always offer; and thirdly, that, however mysterious and however little we know about it, there seems to be no doubt of the fact that certain abdominal neoplasms have cleared up after such incision, and therefore I think we ought to give patients just this one chance even if it is only one in ten thousand: even if complete disappearance of the mass does not follow, these cases so often show improvement and very considerable improvement after such an operation.

Just another point in nomenclature is worth consideration. An objection should be raised against the use of the term hypertrophic, so commonly applied to large alcoholic livers. Believing as I do and asserting unhesitatingly that in fully two thirds of the cases of ordinary alcoholic cirrhosis the liver is enlarged, I cannot see that we can gain anything by speaking of them as hypertrophied,—I should like to hear them spoken of as simply large cirrhotic livers. True hypertrophic cirrhosis as described by Charcot is an exceedingly rare disease, and met with practically only in children; this view enters into no discussion as to the precise anatomical changes that can be produced in the

liver by alcohol or any other poison, (these changes I have lately had reason to believe may be very varied and perplexing,) but it is simply meant to represent a clinical statement. As regards the size of a cirrhotic liver, it is merely a mathematical question of relative preponderance of two factors, exudation of small round cells which increases the bulk, and contraction of subsequently formed fibrous tissue which diminishes the bulk; and in fully two thirds of the cases certainly the former process has the upper hand, and the livers may reach down to the umbilicus and sometimes below it.

Phthisis and the Creasote Treatment.

In this case Dr. Coghill's method of hypodermic injection of beechwood creasote was carried out, but has been discontinued. I have recently tried the plan in two cases in the wards; one was a man of 30, and the other was a boy of under 20. The first was a chronic case: I began by injecting 1*m* twice a day, and pushed it up to 12*m* twice a day, but I have not been able to convince myself that, after a few months' treatment, any satisfactory improvement has taken place; the man lost his appetite and certainly seemed to drift downhill. On the boy, an acute case, the creasote seemed to have no effect at all. In this case we are now giving it by inhalation and by pill, and certainly the temperature has come down, and the cough has been much improved. Ten drops of creasote and the same quantity of chloroform are given on an inhaler constantly. The poor appetite of this patient has, however, not much improved.

Diagnosis of Suppuration in Gonorrhœal Rheumatism.

The next case is of interest as showing how mistakes can be made in medicine, in the diagnosis of pus. It is the case of a girl who in March last began to be ill with rheumatism, which speedily confined itself to the right knee and elbow; there were sufficient pus cells in the urine to make a diagnosis of gonorrhœal rheumatism fairly certain. The knee after some time became so red and angry and fluctuating that everyone who saw it felt certain that pus was there. Accordingly a surgeon was called in to open it; no pus was found, but some dirty-looking serum was let out. The immediate results were not particularly brilliant. After the

operation the temperature did not improve, and the patient was apparently going downhill until she came back to the medical wards, when her temperature immediately fell and she has been going on well since ; the treatment has been quinine and iron, and above all, rest. The point is that as a matter of fact and experience pus never is found in gonorrhœal rheumatism, however strong may be the indications of suppuration in the joints.

Hemiplegia and Pregnancy.

The history is that two days after confinement this patient got out of bed, told her mother she was going to have a fit, had a sensation of pins and needles all over her, but particularly on the left side, and then she noticed loss of power in left side. This took in all about 2 hours ; the face was not affected, and she did not lose consciousness. She has made practically a complete recovery, and I cannot bring myself to believe that the condition was really due to a haemorrhage. I believe that if you get a haemorrhage causing a definite, a really definite hemiplegia, that the recovery will never be quite perfect. Therefore, if you get a so-called hemiplegia with a *complete* recovery, it immediately excludes haemorrhage as the cause—that is the view which I take of this case myself ; the cause was probably either mere spasm of an artery or venous thrombosis, and one might speculate on the excess of fibrin that is said to be present in the blood of women in the puerperium.

This patient further complains of pain in her side, and it may be one of those cases of old diaphragmatic pleurisy with adhesions. In about three autopsies out of five on *adults* these diaphragmatic adhesions are found, and I believe them to be the explanation of the obscure pains in the sides of the thorax so often complained of by patients ; they give rise to no physical signs, and are usually attributed vaguely to dyspepsia.

THURSDAY CONSULTATION AT ST. BARTHOLOMEW'S HOSPITAL.

Sarcoma or Inflamed Veins.

Mr. BOWLEY : In this patient the condition is very easily seen ; there is a swelling with discolouration of the skin over it, commencing at

the inner side of the left tibia and extending to about its middle third. The swelling is lumpy, the skin is shiny and red ; the swelling is elastic, and is fixed to the subjacent tibia. The man is 58 years of age, and he was sent up two days ago. The patient's statement is that twelve years ago he injured the left leg and was laid up for two or three days ; he noticed a lump there three or four days after the accident, and then he asserts that he has had this lump ever since, but a month ago it began to pain, and has since got larger. There are enlarged glands in the left axilla as well as in the groin ; the largest glands are in the left groin, but there are some in the right groin. This patient, on being sent up here, was told that he would have to lose his leg. The question now is, what is the nature of the swelling on the tibia ? There are points about it that make it look like an inflammatory swelling. If his story can be taken as true it can scarcely be a malignant case, because of the twelve years' history, and the way in which the swelling extends down along the shin bone and the reddening of the skin almost suggests inflammation there. It is just possible that it might be a mass of inflamed veins with clotting of blood ; the other alternative is sarcoma, and I am afraid that that is what it will turn out to be. I did not think the diagnosis was sufficiently clear, for he had only been under observation for 14 days, under a medical man outside the hospital ; therefore delay, in my opinion, is advisable in this case. I think the enlargement of the glands in several parts of the body rather discounts the enlargement of glands in the groin ; it might be suggested that these glands are from new growth, but I am not disposed to lay much stress on the glands being enlarged, their condition might not be associated with the leg at all. My advice would be not to hurry, but to let the man rest in bed, and then, if there is no improvement, cut into the swelling and see what it is ; if it is sarcomatous then I should advise amputation of the thigh.

Mr. THOMAS SMITH : I think that the case is probably sarcomatous ; I consider that the man ought to be kept under observation. In the event of it turning out to be a sarcoma, considering how very markedly the glands in the groin are affected I should feel inclined to remove the swelling locally, and also remove any glands.

Mr. LOCKWOOD: Whatever he may have now, there is one thing which is I think pretty certain, and that is that the patient has had phlebitis of the internal saphenous. The oedematous condition and the pigmentation incline me to that view. With regard to the mass itself I can hardly think that it will prove to be malignant, for although it is very large you can move it quite easily. I think it will prove to be an unusually large mass of inflamed veins containing hard and soft clot. I should not myself wait, but I should find out at once what it is by an exploratory incision, for in any case the removal would benefit, and I should attempt a local removal first.

Aneurism.

Mr. BOWLY: This patient I now introduce is at. 56. I have brought him in here for a swelling on the foot, and as you see, as soon as I put my finger on it the pulsation in the swelling, which is forcible and strong, is stopped; it is situated, as you see, on the dorsum of the left foot, close to the annular ligament, and a little to the outer side. The history is that two years ago an earthenware pipe fell on this man's foot; it swelled a great deal, but the swelling gradually subsided after a few weeks, and he connects this present swelling with the old trouble in the ankle. He does not think that there is any increase in the size since he first noticed it two months ago. I think it presents all the characteristics of an aneurism; there is a pulsating tumour with expansile pulsation, pressure stops it and causes diminished pulsation, and on lessening the pressure on the artery the swelling becomes tense and recommences to pulsate and increases in size. It is unusual to have an aneurism in that spot. It does not seem in the line of the dorsalis pedis artery, in regard to which its position is outside. There may be an abnormal dorsalis pedis artery. Considering that it has been a long time there, and that it is more likely to get larger than smaller, I should propose to ligature the anterior tibial; but the man is not a favourable subject for any operation, and I wish to hear the opinions of my colleagues.

Mr. THOMAS SMITH: I quite agree with the diagnosis, and the treatment should, I think, be what is suggested—that it should be ligatured.

Mr. LANGTON: I do not think that this aneurism is from the dorsalis pedis artery, nor do

I think it is from the anterior tibial artery; I think it is more likely to be on some other branch or the anterior peroneal artery. I should deal with it by Antyllus' operation or by removing it altogether, or simply be content with the operation you propose of ligaturing. I should be inclined to be cautious in the matter, as it may be fed from other branches.

Mr. LOCKWOOD: I think myself it may be formed by the anterior peroneal artery through its communications with the tarsal artery. Sir William Savory operated on an aneurism like this, tying the artery, the result being a complete and speedy cure. I should proceed in the same way.

Mr. BOWLY: I shall tie the artery, and if that is not sufficient I shall proceed to removal.

Pleuritic Effusion in Hepatic Cirrhosis.— The presence of pleuritic effusion on the right side only in Laënnec's atrophic cirrhosis has been noted as rather an exceptional symptom. G. Vallani, however, is inclined to regard it as a constant symptom, having found it in nine cases of cirrhosis examined in Capazzi's clinic. He believes it to be of value in the diagnosis of doubtful cases in which it is difficult to determine whether ascites is due to cirrhosis of the liver, to thrombosis of the portal vein, or to compression of that vessel by tumours or swollen glands. In the latter conditions perihepatitis does not exist, while in atrophic cirrhosis its presence explains the pleurisy by propagation to the right pleura.—*Universal Medical Journal.*

Varicocele.—Dr. J. Brault, of Lyons, has recently performed a new operation in two complicated cases of varicocele, having previously tested the method on the cadaver. He removes a large elliptical portion of skin from the external and posterior surface of the part of the scrotum involved, turning the flap upward and downward; he then exposes the large veins and resects them separately between two ligatures, closing the wound by approximating the flaps and stitching the margins together. He claims that this method is superior to that by which a part of the scrotum is removed through a transverse incision, while it can be rapidly performed, and is accompanied by little haemorrhage. The method is suitable for hydrocele combined with varicocele or with distension of the scrotum.—*Lyon Médical.*

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A CLINICAL LECTURE

ON

WEAK SCARS AND HERNIÆ FOLLOWING ABDOMINAL SECTIONS.

Delivered at University College Hospital, June 13, 1896,

By ARTHUR E. BARKER, F.R.C.S.,

Surgeon to the Hospital, and Professor of Principles and Practice of Surgery and of Clinical Surgery at the Hospital.

GENTLEMEN,—I want to speak to you very briefly to-day about the possibility of weak scars following abdominal operations. With all our modes of procedure we have to face the disadvantage that under certain conditions of abdominal section weak scars are certain to result from our operations, no matter what precautions we take, and under other conditions are very likely to follow unless we adopt certain measures which I venture to think are too often neglected. This liability would not in any way prevent our doing an operation in an emergency, for then the risk of such a defect must be faced, our first duty being to save life. If a weak cicatrix should happen to follow, the patient must try to bear it with the help of appliances, or else submit to a subsequent operation with the object of bringing about some improvement. But it is important to consider whether, in any given case of abdominal section, we cannot adopt measures which shall prevent the formation of weak scars, and so obviate the necessity of a secondary operation, perhaps years afterwards, to prevent the occurrence of a ventral hernia.

We must first consider the conditions which determine the formation of a weak scar in the abdomen.

There are, roughly speaking, two classes of cases which we have to deal with here, namely (*a*) cases in which we operate for suppurative conditions, and which require drainage; and (*b*) cases in which we operate for conditions not associated

with suppuration, or where suppuration can, by the exercise of care, be limited or entirely prevented. In the suppurative case, the process of repair in the abdominal wall is effected by the production of a considerable amount of imperfectly formed fibrous tissue, which is badly vascularized and weak. In cases of primary union, in our incision, on the other hand, there is a minimum of fibrous tissue, and that material is highly organised and very strong. That is the key-note of the whole matter.

Let us now take cases in which suppuration is present at the time of operation, such as a case of gangrenous appendicitis, of which I hoped to show you an example to-day, and see what happens there. You have already seen the patient I refer to, but he has just left hospital. The conditions under which that man was originally operated upon, and those which followed the operation referred to, made it practically impossible to prevent the formation of a very weak scar. He is 22 years of age, and came in here a year and a half ago with the worst attack of appendicitis I ever saw. I operated at once, and removed the sloughing appendix. Then, contrary to a practice which is almost universal, and following a line which is, at all events, in certain cases justifiable, I attempted in the first instance to close the wound and do without drainage, although the wound was a suppurating one, so anxious was I that the man should have a sound scar. My own feeling is that there are a certain number of suppurating cases in which you can take away the appendix, clear out the pus from the cavity, dry it out thoroughly, and dust with iodoform, and close by suture, and by this means secure primary union. In this case, however, as the attack was of a gangrenous character, and suppuration went on around the original seat and in the cavity where the appendix had lain, I had to subsequently remove one or two of the stitches, and put in a very large drainage-tube. This drained it, the man got quite well, the opening closed, he went away to a convalescent home, and came back strong. But the scar was weak, not only as regards the skin, but in the

abdominal parieties, which did not unite, having been attacked by the same gangrenous process which destroyed the appendix. Now, under such circumstances the scar must necessarily be weak; first, we failed to keep the edges together; secondly, the edges had sloughed, therefore there was a wider patch to be filled up; thirdly, suppuration went on destroying the formative material as fast as it was thrown out, and what was ultimately formed was weak and became badly vascularized, consequently ready to yield to pressure from within. Therefore, I was not surprised to find him coming a year and a half afterwards with the scar stretched wide and very thin, and with a commencing ventral hernia.

We will take another case. A woman now in the hospital was here eight years ago. She came to me with hydatids in the liver. I made a median incision below the ensiform cartilage nearly to the umbilicus. I found that a cyst in the liver was full of hydatids. In those days we were a little more shy of tackling cavities in the liver without first securing adhesion between the latter and the parietal peritoneum than we would be now. The course I adopted was to pack the wound with antiseptic gauze, and allow an adhesion to form between the liver and the peritoneum before I actually opened the cyst. This packing kept the edges of the wound, of course, wide apart. Having stripped the parietal peritoneum off for some distance round the edges of the incision, I pressed it down upon the liver, and it united there. But after four or five days, when I took out this packing of gauze and found the liver adherent everywhere to the parietal peritoneum, it was plain that the edges of the wound had never come together; there was nothing to hold them so. I incised the liver, and took out the hydatids, and drained the cavity. The patient got well, but with the edges of the rectus wide apart. There was no suppuration, but that interval between the muscles had to be filled up with granulation tissue. The patient came up here a few weeks ago with a bulging scar and a hernia of the transverse colon; these it became necessary to deal with.

Then there is the case of a wound which has apparently healed by first intention, yet there is a tendency to a very weak scar. That may take place in various ways, but the commonest is the

following:—You have operated on a condition which does not necessarily involve subsequent drainage, and you have proceeded to stitch up the abdominal wall. There are various ways of doing this, but in the case in point you probably have not included sufficient of the muscles on either side in your stitches. Possibly you have taken up the skin very carefully as well as the peritoneum, but not enough of the true wall of the abdomen; some of the fascia has been caught up in your stitches, but the proper sheath of the muscle has not been included. The wound appears to unite by first intention, and you think a sound scar will result. Yet in a year or two you find that it has stretched, and if you come to operate on a case of that kind you will find evidence that in the original operation you had not included sufficient of the deeper structures in the stitches. On the other hand, I have lately shown you some patients where seven or eight years after abdominal section there has been no trace of a bulging scar. Those are the cases where there has been not only primary union, but where the structures involved have been carefully applied one to the other over a large area, and not only the edges of them but the body of the material. That leads us to the consideration of what has to be done in these several cases.

Where you must drain on account of septic suppuration, drainage is the first consideration—all questions about the weakness of the scar must be put aside as secondary. I do not say that every suppurating abdominal wound must necessarily be drained, because I have opened the abdomen in many cases, taken away, for instance, a suppurating appendix, and obtained primary union; also in tubercular peritonitis, as well as in other forms of inflammation of the peritoneum. But where it is obvious that there is septic pus accumulating, you must provide, by drainage, for the free escape of this septic material, leaving all other questions for the future.

Then when you are going to attempt closure of the abdominal wall, important questions arise which are not yet settled. Having recognised the fact that in a great many of the cases where a weak scar has formed, it has really been a question of want of proper apposition between the various layers in the abdominal wall, surgeons have begun to bring these together with a great deal more care than was formerly considered necessary.

Two or three quite distinct ways are adopted. Many surgeons, in closing an abdominal wound, begin by carefully stitching together the edges of the peritoneum, then they put in a row of sutures in the borders of the muscular tissues, and draw these edges separately together ; some even stitch two or three layers separately and leave these sutures *in situ*, and close the rest of the wound by a fourth row of superficial sutures. Others, again, are quite content to take muscle and peritoneum together with one row of sutures, and close the skin over that. Others—and I confess myself one of them—believe that if the sutures are put properly through the skin, through the muscles, and through the peritoneum, only one row of sutures is required, which can subsequently be removed. I have tried all these methods, otherwise I should not consider myself competent to express an opinion upon them. Some, who are entitled to our respect, believe that the scar is less likely to be weak after two or three rows of deep sutures. I have tried them, but have come round to the belief that one row of stitches to include skin, muscle, and peritoneum is the best for all ordinary purposes. The great objection, to my mind, to the use of a treble row of buried sutures is that a great deal more time is occupied, and time is a most important consideration in all abdominal operations, indeed one of the most important. The next point is, that if any of these stitches suppurate subsequently,—and this is a very possible contingency, no matter what care has been taken—then the skin often unites over this suppurating area. This subcutaneous abscess, then, before it is opened, may cause the material which has been deposited in the wound to become loosened, and thus produce a weak bond of union between the two edges. Finally, I do not myself think that in ordinary cases there is more likelihood of the edges being satisfactorily brought together by that treble row than by a smaller number of deep inclusive sutures, that is as regards catgut, silk, or horse-hair. But when one comes to use silver wire I think the case is a little different. When operating on a case which I expect not to suppurate, I now act as follows, and if you keep these points in mind you will have very little weakness in your scars. When you are bringing the stitches together, say in the middle line, you must take up the skin very near to the edge of the wound, and put the

point of the needle through it not more than a quarter of an inch from the edge of the wound. With the skin now drawn back, see that your needle goes well into the rectus on either side, at least half or two thirds of an inch, and that it takes up both layers of the rectus sheath—anterior and posterior. The finger of the operator will, of course, be in the abdomen, and care must be taken that the needle takes only the peritoneum on its edge—not a great wisp of it—and does not turn the peritoneum out between the edges of the rectal aponeurosis. If this course be followed, the cut borders of the peritoneum are brought just edge to edge, and no more, while the rectus is brought widely in contact, and the skin only just together.

One frequently has seen that in putting in stitches in the abdominal wall they have been put in a good way from the edges of the wound in the skin ; then they have gone obliquely only just through the edge of the muscle. Sometimes the stitches have missed the anterior or posterior layer of the sheath, and have taken up no muscular fibres, but have included a large strip of peritoneum, which is usually loose, and when the stitches have been brought together the peritoneum has been folded and puckered, and turned outwards between the other structures of the wound ; consequently, the parts which are essential to the strength of the abdominal wall have not been approximated and included in the stitching. And you must remember that the adhesion which is between the peritoneal surfaces, although it occurs very readily, is, in the nature of things, an adhesion which very soon disappears. The lymph which is thrown out in aseptic cases is just enough to glue the surfaces together, and after a time is absorbed almost completely. You see that if you open an abdomen which has been the seat of inflammation years before, here you do not find any adhesions in many cases. If over the edge of the incision there is a quantity of peritoneum actually coming out, and skin intervening between the muscular layers, the adhesion of these surfaces is good for the time being ; but after a while it becomes absorbed, the parts unfold again, and there is only skin and peritoneum united—nothing else. As a matter of fact, the peritoneum should be folded inwards and the muscle edges firmly united together.

When you are doing an operation which goes

through the external and internal oblique, and transversalis, you have to be particularly careful about these points, because there is a tendency for the sheaths and muscular fibres to retract considerably. You are apt to leave the muscles entirely out of the stitches, consequently there is only peritoneum and skin united in the ultimate scar.

Another point which, I think, plays a very important part in the strength of the scar is the inclusion of muscle-fibres in the stitches. I consider this so important that, where I have a choice, the position of my incision is determined by the presence of muscle rather than of an aponeurotic line, because in the former case the scar is far firmer. When you do operate through the muscle, remember that when you have once divided the skin and just touched the anterior aponeurosis of the muscle, you should never cut but merely separate the fibres. That was first drawn attention to by, I think, an American surgeon. When the uncut muscle fibres resume their old place, the abdominal wall is left practically as solid as before.

Therefore, to sum up, do all you can to prevent suppuration. If that is impossible, see that the suppuration is as limited in time and extent as it can be made by proper drainage, leaving at least the middle of the wound open. Then, when there has been only slight central suppuration as in some cases of tubercular and other forms of peritonitis, you may trust to clearing out the cavity of the peritoneum without drainage at all. For we know there are cases in which the appendix inflames and suppurates, which are never operated on at all. What becomes of the pus then? The peritoneum is capable of disposing of it in some way, therefore we are justified in expecting the same to occur after operation in certain cases; where we have opened the abdomen and cleansed the cavity we leave less to be disposed of by nature than in many cases has been so disposed of by the natural processes when no operation has been done. Where there is no suppuration to begin with, we must most strenuously endeavour to ensure that the scar shall not suppurate, which is possible in most cases. Then if, subsequently, a weak scar has formed, it becomes a question of radical cure of a ventral hernia. That leads me to draw your attention to a detail of great importance.

In dealing with a ventral hernia following upon such a scar, you have a choice of the three methods of closing the abdomen to which I have alluded. Which of those should you chose in operating for the radical cure of a ventral hernia? I have tried various ways, and I now adopt a middle course in these cases. I let the peritoneum take care of itself; that is to say, bring it edge to edge without including it in the stitches, or you may close it by just a row of silk or catgut, and then put in a row of silver sutures passed as thickly as possible, through the proper muscular tissue of the edges of the wound—the rectus in one, and external and internal oblique and transversalis in the other. I pass the silver sutures rather closely together, not only to bring the edges very firmly together by material which excites very little irritation, but to ensure that that material itself shall form a kind of barrier against the descent of hernia. In the two cases I speak of I proceeded as follows: Having freshened the edges of the abdominal wound, and cut out the weak tissue which intervened, I held the edges of the rectus and the skin and passed silver sutures very deeply and far from the edge. Those stitches included every bit of the abdominal wall minus the skin and peritoneum, and the stitches were about a quarter of an inch apart. When I began to twist the stitches and draw the edges together, I inverted the edges in such a way as to bring the outer surface of the rectus back to back, and pushed the edges towards the abdominal wall. In that way I have got a very large surface between the edges of the rectus on both sides, and have presumably got that to adhere, for both cases have healed perfectly *per primam*. But, besides, there is a row of silver sutures in the abdominal wall, like a gridiron, so that at every quarter of an inch there is a fine silver band surrounded by fibrous tissue, forming a very considerable barrier to the re-descent of the hernia. Silver wire gives no trouble, and very effectually ensures the ends we have in view.

Deep silver stitches are, of course, meant to remain in the abdominal wall permanently; therefore you must use the very best material obtainable, and it must be well tempered, otherwise the wire will break, and the sharp points will stick in at various places, and perhaps give trouble by penetrating the peritoneum. You should make the

wire red-hot, and then allow it to cool slowly. Let me warn you against drawing the wire through your fingers, or hammering it, or stretching it in a vice before use. These processes help to make it brittle. After twisting the wire, cut it off about a quarter of an inch from the stitches, and be careful to turn in the cut ends so that they shall not stick up against the skin; then there will be no unpleasant sensations.

I wired a boy's clavicle some years ago, and he came to me subsequently complaining that he could not wear his brace. I found a point of wire standing out under the skin, and I turned it down, with complete relief. A similar event may occur in the abdomen.

If you examine the two patients to whom I have referred, I think you will find the scars very sound, and likely to remain so, as was certainly the case in other cases similarly dealt with which I have seen long after operation. The remarks I have made on the necessity of including a large quantity of muscular tissue in the stitches when closing the wound apply also to operations for the radical cure of other forms of hernia. No radical cure will be obtained without this precaution; whereas, if you follow the advice I have given you, the resulting scars will resist any strain which is likely to be put upon them.

DEFECTIVE METABOLISM IN ITS RELATION TO GOUT.

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THAT the normal metabolism of the human body, both as regards tissue change and the elaboration of the alimentary substances introduced from without to maintain them, should produce no disturbing effects on the organism of a healthy individual, we regard as the first principles of physiology.

By a perfect metabolism we mean a perfect functioning of all the various physiological processes that take place in the body. For convenience

of classification we group these processes under anatomical heads called systems, all inter-dependent on each other in maintaining the health of the individual, and derangement in any one of which is followed by imperfect functioning of the others, producing evidences of defective tissue change, both destructive and constructive—all of which the term metabolism comprehensively includes.

In this paper I am only concerned with those systems that seem to me to be mainly responsible for the production of a vicious change in this metabolism, leading to what we understand as uricacidæmia, or gout. According to the most recent views gout is uric acid in excess, which, driven from the blood in a sub-alkaline condition of that menstruum—being no longer held by it in solution—precipitates itself in back waters, so to speak, of that fluid where the stream is slowest and weakest, carrying with it in its flight some of the soda salts of the blood; and, after precipitation, robbing the tissues around it in its new resting-place of all alkaline salts contained in them, to return again to the blood when a rising alkalinity of that fluid tempts it back, or to remain in its resting-place if a sub-alkaline condition of the blood continues. Both in the blood and out of it in the tissues, it is, when beyond a certain proportion, a foreign body, and manifests itself in both situations by well-marked symptoms. All recent writers on the subject are nearly agreed as to this being the condition of affairs in gout; but what they are not agreed on is, as to why uric acid in excess should be present in the blood?—and it is on this aspect of the question I venture to offer some remarks.

Whence comes this excess of uric acid? According to those who regard it from a merely chemical point of view, the excess is due to the food ingested being too highly nitrogenous, and the blood—being low in alkalinity from acids introduced with them—is an insufficient solvent for uric acid formed out of the compounds of urea at or in the renal barrier, and the kidneys failing to eliminate, it is carried back again into the circulation by the renal veins. Whilst others explain it by the individual possessing a hereditary tendency to form uric acid in excess, and the kidneys failing in elimination, it gradually accumulates in the blood. These are the chemical views of the aetiology of gout, with the corollary deduced from

them as to its rational treatment from a purely chemical point of view of giving alkalies, or alkaline salts, that render the blood more alkaline, and are good solvents of uric acid, and behold ! the uric acid is eliminated by the kidneys, and appears in increased quantity in the urine. This simple view of the matter has opened the gates to all kinds of quackery and quack remedies, all claiming to be solvents of uric acid ; and the professional, as well as the public, mind is captivated by the brilliant results that have been said to follow the exhibition of such remedies. But after a little the gouty symptoms return again, and then the thoughtful mind turns to consider what other causes than mere chemical ones will explain this vicious recurrence. Nearly every writer on the subject—since Garrod discovered uric acid in the blood of the gouty—has put forward theories to account for its presence, and its over-production. Imperfect oxidation of urates, put forward by Murchison, and advocated by Garrod and later writers, certainly seemed to take hold of the professional mind, and has to-day many strong advocates. It has certainly, in theory at all events, chemistry to recommend it. Lately Harbaczewski, from chemical investigations, has brought forward the view that uric acid is the metabolic product of nuclein derived from leucocytes. Its increase in the blood and urine in leucocythaemia, and in pernicious as well as in other forms of anaemia, lends strong support to his views. All cases of gout are, however, not associated with symptoms of either leukæmia or pernicious anaemia, though a transient anaemia is very frequently associated with the first symptoms of an excess of uric acid in the blood, and one form of gout—that known as atonic gout, occurring in the flabby and lymphatic—is characterised by anaemia as a marked symptom ; but to this I will allude later. Harbaczewski's theory as to the source from which this excess of uric acid is derived applies to only one of the great physiological systems—the circulatory one ; but, in my opinion, defective metabolism in others exercises from time to time a preponderating influence in this excess, and it is with a view of calling attention to this that my remarks principally apply. As far as my views have led me along this line of observation and investigation, it has, I think, a very important bearing on the question of treatment of uricacidæmia and gout, for we cannot—looking at

the matter from the mere chemical standpoint alone—hope to rid our patient of this vicious tendency by merely dissolving out uric acid from his system if we leave the causes that are constantly adding to it untouched. What these causes arise from will require a review of all the various physiological processes of the body included in the system groups I have mentioned, and defective metabolism in which adds to the blood the various compounds of urea in such quantities that the excretory organs, especially the kidneys, cannot deal with it, and in their efforts to do so form from the light soluble urea compounds the heavier and more concentrated uric acid. This defect, as we all know, increases with age as the tissue metabolism becomes less and less active, and its first onset is coincident with this lessening of metabolic activity. The views put forward by Murchison as to lithæmia being the forerunner of gout seem to me to afford strong evidence in this direction. Lithæmia is but the first evidence of lessened metabolic activity ; and as age increases, this lessened activity increases with it, until such a condition is reached that one of the most simple metabolic processes in the body—the conversion of proteids and waste products into urea—becomes a most complicated one, leading to a gradual auto-intoxication, having lithæmia at one end in its primary stage, and uric acid at the other in its final one. That this metabolic defect is not confined to one function or one system alone I am firmly convinced, and though the digestive system is singled out by writers of to-day as the chief sinner—and in the majority of cases with very good reason—it is not the only one. I shall take up the various systems in the order of their importance, and begin with

THE NERVOUS SYSTEM

first, as it dominates and overshadows every physiological and pathological process of the body. Its influence in the production of gout is invoked by every writer on the subject since the time of Sydenham, and in his Treatise on Gout Sir Dyce Duckworth lays stress on the undoubted influence exercised by the nervous system in its production. Many other writers do the same, including Dr. Ralfe. Sir Dyce Duckworth regards gout as a neurosis, which descends as a hereditary legacy from progenitors. Given the same conditions in

two individuals to form uric acid in excess, the individual with strongly-inherited neurotic tendency will develop gout, and the other will not. We know in other neurotic affections that the neurotic tendency is a transmitted or inherited one, and it to some extent explains how the liability to gout in families becomes hereditary, from an innate defect in nervous metabolism transmitted from one generation to another. That this neurotic element is strongly marked in gouty families we cannot deny, and that gout alternates with other neuroses in such subjects is equally well known. The descendants of a gouty stock, though they may not themselves show evidences of uricacidæmia, are the victims of other neuroses—such as epilepsy, hysteria, hypochondriasis, megrim, insanity, insomnia, and neuritis, with many other nervous affections.

Nor is this instability of the nervous system confined to those who inherit the gouty tendency, for in those who acquire it by indulgence we have evidence of this instability. Such individuals possess a nervous system that is (to use a popular expression) "easily upset;" they are of an anxious and sensitive disposition, choleric and irritable, easily cast down and easily excited by trifles, with evidences that point to vaso-motor equilibrium being easily deranged, and a tendency to flush and sweat from slight causes. Amongst women such instability is evidenced by hysteria, dissatisfaction, and emotion, morbid nervous energy, with palpitation and ovarian irritability, and, at the menopause, a tendency to involuntary flushing and tachycardia, with or without symptoms of Graves's disease. In fact, the gouty individual possesses a vulnerable nervous system, which is not confined to one portion of that system alone, as the higher centres, as well as the vaso-motor and trophic ones, share in it.

How will these evidences of altered metabolism on the part of the nervous system produce an increase in the blood of morbid material, out of which uric acid is formed? By the increased disintegration of nervous elements, which must accompany all perverted over-action in any part of that system, we cannot originate a single mental conception, nor produce a volitional impulse, the result of that conception, without causing a change in the metabolism of the cortical cells of the brain, and this extends to all parts of the nervous tracts that take part in it. We are all familiar with the

causes that directly produce or originate gouty attacks from mental conditions in the predisposed, such as over-work of brain, anxiety, exhaustion of brain and nervous system, loss of sleep, and shock from any cause. Even in many diseases of the brain, accompanied by increased disintegration of brain cells, uric acid and gout are frequent accompaniments; and our lunatic asylums furnish abundant examples of such conditions. The blood is flooded by the products of such nervous metabolism, or rather defective metabolism; and when the nervous regulating mechanism, that presides over their conversion into simpler compounds to assist in their elimination, is in an unstable condition they accumulate in it.

Nor is evidence wanting on the physiological and chemical side to show this, as the following substances have been found in nerve tissue associated with defective metabolism—xanthin, hypoxanthin, and kreatin, substances closely allied to or easily converted into uric acid.

THE DIGESTIVE SYSTEM.

The system which comes next in importance, from my point of view, as a factor in this imperfect metabolism is the digestive system. Indeed, it may be broadly stated that in the acquired form of gout it plays the *role* of chief factor, and, according to Dr. Haig, the only one in the production of uricacidæmia. We have to thank this observer for the side-lights he has thrown on the pathology of uric acid, and the patient labour he has bestowed in experiments on himself to show the dietetic causes which produce an excess of it in the blood, and for the ingenious theories he has brought forward to explain its behaviour and its various effects, both in the blood and out of it, many of which, supported by experiments, appear to be reliable. He has established and confirmed the important fact that uric acid exists in the blood in a definite ratio to urea, which in health is about 1 to 35, and that any increase of it above this proportion constitutes a condition of uricacidæmia, its future behaviour as to elimination, or increase, being altogether regulated by the alkaline or sub-alkaline reaction of the blood, and by additions from the dietetic side. He has added to and confirmed the valuable investigations into the chemistry of uric acid made by Sir A. Garrod, Sir Wm. Roberts, and Professor Latham.

Whilst I fully agree with him that the chief causes for the excess of uric acid come from the digestive side (due to faulty metabolism in the splitting up of nitrogenous compounds), I cannot agree with him in the views he holds, that the derangements in the various parts of the body (the digestive tract included) are but the results of the presence of uric acid present or precipitated in them, and not the cause of its production.

From my point of view, embarrassment of function in any part of the digestive tract, arising from dietetic excesses in the first instance, or to gradual lessening of functional activity as age advances, or to changes in the normal secretion of the glands that take part in it, the result of disease or derangement, is the cause of this defective metabolism, which leads ultimately to the plus production of uric acid. Let me take up the first cause I have alluded to—dietetic excess—in its bearings on the subject. We all know from observation how long dietetic excesses may be indulged in by the young, and also by the healthy adult, whilst the fierce fires of a healthy metabolism are unchecked by derangement or disease. As long as the digestive organs are healthy the only result that follows the excess is an increased quantity of lithates, or urates, deposited in the urine from a temporary embarrassment, but the perfect metabolism soon returns to its normal condition, and no ill results follow, and this condition may go on for years; but let a catarrh of the stomach arise from any cause leading to fermentation in the gastric contents and the formation of fatty acids, and then the first evidences of a defective metabolism in nitrogenous compounds commence, increased gastric acidity with lowering of blood alkalinity from their absorption follows, and uric acid begins to manifest its presence. With the disappearance of the gastric catarrh a healthy metabolism again asserts itself, urea is excreted in normal quantity, and uric acid falls, not to return again until a fresh gastric catarrh supervenes. If the individual is now nearing the period when the general metabolism is beginning to fail, and its fires to burn lower (which in many individuals takes place towards the end of the "thirties," but may be delayed to the "fifties" or even "sixties"), he realises, as the popular phrase puts it, that "he has a stomach

or a liver," which may be translated into—his metabolism is failing, and uric acid is being constantly added to his blood. If from alcoholic or dietetic excesses, his stomach catarrh now becomes permanent, or his liver functions deranged from the same cause, the picture is complete. There is no longer a healthy rebound in his metabolism, his waste products accumulate, and uricacidæmia is established, and a trivial accident, or lowered nervous influence—the result of some illness—may determine precipitation in his joints.

Though this defective metabolism may have its origin in different portions of the digestive system, I am satisfied the stomach is by far the most frequent situation of its inception. The various derangements of this organ, associated with fermentative changes, and lessened or altered secretion with dilatation, and the formation of fatty acids, and of toxins, the result of microbial growth, must all contribute in lowering its metabolic activity. It is well known to the observant physician how frequently dyspepsias arising from these causes precede for years the advent of the gouty manifestation; and he can generally foretell its ultimate appearance from their persistence. Persistent acidity is a well-known symptom in those who ultimately become gouty.

That the liver plays a most important part in the metabolism of nitrogenous compounds there can be no doubt, and that its derangement is followed by a lessening of this metabolism we must all admit.

It is not necessary I should allude to the physiological evidences in support of these facts, as the literature of the subject has established them; but I may again allude to the observations of Murchison in his Croonian Lectures on "Functional Derangements of the Liver," where he points out that lithiasis is the result of a faulty metabolism of that organ, due to functional derangement; that this symptom occurring in children generally indicated a transmitted tendency to imperfect function on the part of the liver; and that the subjects of it were generally the children of gouty parents. I may also allude to the chemical investigations of Prof. Latham, in connection with defective metabolism on the part of the liver, where he states: "Just as in diabetes the essentia

fault lies in the inability of the system, either in the liver, or it may be elsewhere, to effect the metabolism of glucose, which then passes into the circulation, and is discharged by the kidneys, so in gout or gravel the imperfect metabolism of glycocin, a derivative of glycocholic acid, is the primary and essential defect. Unchanged, it passes from the alimentary canal, or elsewhere, into the liver; there, under the action of the gland, it is conjugated with urea, resulting from the metabolism of the other amido bodies, leucin-tyrosin, &c., and is converted into hydantoin, or a kindred body, then passes on to the kidneys, to be combined there with another molecule of urea—forming ammonium urate—a portion of which overflows into the circulation, and is converted into sodium urate." Besides functional derangements of the liver inducing a defect in its metabolism, it is a matter of observation how frequently catarrhal congestion of that organ precedes the actual manifestation of gout, and an arrest of the secretion of bile, with clay-coloured stools, often precedes an attack. Ebstein has recorded cases of hypertrophic cirrhosis in gouty men even without portal-venous obstruction; and Murchison noted the frequency of cirrhosis in connection with gout, and remarked that "the condition of liver which develops gout renders it liable to suffer from alcohol, even in small quantity;" and Sir Dyce Duckworth observes—"The liver is the organ in which in health uric acid is chiefly formed, and it is probably to derangement of function in this gland that we must look for over-production of this substance."

Another portion of the digestive system which contributes a very considerable element in this defective metabolism is the intestine; here, as in the stomach and liver, we have constant evidences of it—associated with or preceding gout. Constipation from inanition or atonic muscular action is constantly found, alternating with fermentation of its contents and diarrhoea, and a proneness to catarrh from slight causes.

The researches of Bouchard in his work, "Auto-intoxication," have abundantly proved the many toxins which may be absorbed from the intestine. Most of these toxins, the result of microbes, many of which are normal inhabitants of the intestine, may, owing to lessened metabolic

activity on the part of the excretory glands (especially the liver), obtain a temporary mastery, and paralyse the intestinal functions. He has also pointed out that the absorbents, from undue delay associated with constipation, abstract many poisonous compounds from the faeces which are hurtful to the economy in their passage through the blood before being excreted by the kidney. His experiments with intestinal antiseptics, and their effects in diminishing the poisonous alkaloids subsequently extracted from the urine, prove this. Kreatin, leucin, and tyrocin, all easily convertible into uric acid, are derived from the intestine during defective metabolism, as Latham has pointed out.

THE CIRCULATORY SYSTEM.

The system which ranks next in importance, after the digestive one, in the production of this imperfect metabolism, is the circulatory system; and, if Harbaczewski's conclusions are correct, it should prove the most important one in the plus production of uric acid in the economy. According to this observer, uric acid is the metabolic product of nuclein derived from leucocytes, and he contends that gout is a leucolysis—a perverted metabolism in blood-formation—and he reasons from the plus production of this acid present in the blood and urine in both leukæmia and pernicious anaemia, a well-known clinical feature in both these diseases. If so, this perverted metabolism is closely connected with both blood-destruction and blood-formation, and the presence of this acid in excess in the spleen and liver in uricacidæmia is to some extent accounted for.

If an increased formation and destruction of leucocytes in this affection takes place, it should certainly diminish their contributory aid to general blood-formation, and lead to a poverty in red corpuscles as well, and so explain the anaemia that marks many of the atonic forms of gout. But there are other portions of the circulatory system which afford evidences of this metabolic defect besides the blood. We find clinical evidences of it through the entire circulatory channels from the heart to the capillaries. Coincident with the first evidences of failure in the general metabolism in adults nearing middle age, comes an atonic condition of the heart muscle itself—its contractions have lost their strong muscular element on auscul-

tation, and the heart becomes more excitable and more easily disturbed, more rapid after exertion, and prone to intermit, and there is a greater tendency to dyspnoea after violent exercise on the part of the individual than formerly. Pathologically there may be found no change in the cardiac muscle, but clinically we recognise that there is some defect evident, either a neuropathic or a nutritive one, affecting its muscular tone. The arteries, and especially the veins and capillaries, seem to share this, and their regulating vaso-motor mechanism seems also more easily disturbed, and their trophic functions to suffer, rendering the coats of the vessels more prone to changes—whilst the arteries from their greater resistance show at first least evidences of this, the veins and capillaries show early signs of it. Defective metabolism in the capillary circulation may, to some extent, explain the trophic changes in the skin that are so frequent in the gouty, and also the failure in elimination of uric acid by the capillaries of the kidneys, to which Garrod and so many other writers allude. Evidences are visible to the naked eye in the colour of the skin when this failure commences—as the florid man begins to look pale or livid, and the pale man sallow or bilious-looking—whilst a tendency to thrombosis occurs in the capillary veins, rendering them prominent. We know how frequently phlebitis ultimately supervenes in the veins of the extremities, preceded by or accompanying thrombosis in the veins of the rectum, leading to haemorrhoids; and also in the veins of the pharynx and larynx, leading to congestion of their mucous membranes.

Dr. Haig would explain the vascular changes by the high arterial tension and contracted capillaries resulting from the presence of uric acid in the circulating blood, and the irritating and poisonous effects it exercises on the coats of the vessels and on their vaso-motor nervous supply. I fully agree with him that in pronounced uricacidæmia the sphygmograph indicates this high arterial tension, but this is quite consistent with a lowered metabolism in the circulatory system generally.

THE LYMPHATIC SYSTEM.

Intimately associated with this lessened metabolic activity in the circulatory system comes a lessened metabolism in the lymph channels, which they subtend, and especially in the lymph spaces, with

lessened oxidation of the waste products contained in them, derived from the metabolism of the tissues generally. In this system we have a fertile source of production for the early progenitors of uric acid, if the heart and circulation is defective. The lymph-corpuscles supply abundance of nuclein, which, according to Harbaczewski, is one of the chief sources of uric acid, and the plasma is found to be rich in leucin and xanthin. Lauder Brunton has pointed out how essential a free movement of the lymph in the lymph channels is to oxidation and metabolism, and what an influence massage and muscular movement exercises over it from the pumping action produced in the lymph-spaces by both forms of exercise. Muscular movements and respiration are almost the only means by which the circulation is carried on in this system, as it has no motor mechanism if we except the *vis-à-tergo* of the heart and arteries, and if these latter are in default, delay and defective metabolism must result if muscular exercise is not taken. From the physiological side, this is an argument in proof of the fact that sedentary life and deficient exercise conduce to uricacidæmia and gout.

THE RESPIRATORY SYSTEM.

I will now allude to the part played by the respiratory system in this defective metabolism. The chief function of the respiratory system is to supply oxygen to the blood, which in its turn supplies it to the tissues, so aiding in metabolism, and in the combustion of waste products contributing to animal heat; at the same time getting rid of CO₂ (a waste product) and of water. If from any cause a defect arises in this system, the process of oxidation is interfered with, CO₂ is retained, and waste products accumulate in the blood and tissues generally. Have we any evidence that defects arise in the respiratory system, and that their effects lead to uricacidæmia and gout? I will take up the evidences afforded, both from the physiological as well as from the clinical side. We know those who live an indoor life, or dwell in bad air, are more prone to form uric acid than those who live out of doors or in pure air. The former get less O into their blood and in addition inhale more CO₂ (a great portion of which is retained), whilst with the latter oxidation is more perfect, and there is no uricacidæmia, though they may have the same causes, either dietetically or by hereditary tendency,

for its production. Or, again, we find there is in some individuals a tendency to greater sensitiveness of the respiratory tract than in others—they catch colds easily, are more sensitive to changes of temperature, prone to laryngeal and bronchial catarrh, or to asthma and emphysema, and these are just the individuals who ultimately become gouty. So frequently is this associated tendency noticed that many pathologists have ascribed gout to the toxins formed by a catarrhal bacillus (not yet discovered) that is supposed to produce laryngeal, pharyngeal, and bronchial catarrh. I think we can explain this liability to catarrhs to an innate defect in respiratory metabolism on the part of the individual, and which is only another evidence of this general metabolic defect, here as elsewhere, though the tendency with many writers is to ascribe the catarrh to the uric acid, and to the precipitation of this peccant material in the affected parts. I am far from denying that we do not occasionally see an attack of bronchitis in the gouty, which is directly due to gouty deposit in the mucous membrane of the bronchi; as uratic deposit has been found there pathologically by competent observers, its presence here would seem to be only accidental, or only part of a general tophaceous deposition, as we know the bronchi are not a channel by which uric acid is eliminated, though it is so for other toxins that directly produce bronchitis-uræmia for instance—and, according to Bouchard, the fatty acids which indicate their presence when being eliminated by this channel, through the peculiar odour of the expired air.

THE MUSCULAR SYSTEM.

The last system I shall refer to in connection with this subject is the muscular system; and here also evidences are not wanting to show the amount of waste products they contributed to the blood both during their healthy metabolism, but more especially when this is defective. Here we have one of the largest factors in the plus production of the urea-forming compounds, which during active exercise is constantly poured into their lymph spaces, and from there into the blood. A glance at the many products of muscle waste will show this—kreatin and kreatinin, xanthin, and hypoxanthin and uric acid. During exertion the muscle becomes acid, and many acids are developed in it—paralactic, lactic, and phosphoric acids—and,

after violent muscular exercise, many of these are retained in the blood, and contribute to raise its acidity, and lessen its solvent action on uric acid. This latter acid was found to be increased in the blood by Handfield Jones, in the person of an Alpine climber, on whom he made experiments after a severe day's mountaineering; but it was found to be afterwards eliminated during the subsequent rest, but not for some time. During muscular exertion very active oxidation of the waste products takes place, O being taken in and CO₂ being given off. But, with violent exercise, the O does not seem to be added in proportion to CO₂, and the latter accumulates in the muscles, giving rise to a sense of fatigue, and lessening their metabolism,—a fact which should not be lost sight of in ordering exercise for the gouty. Moderate exercise increases metabolism, but violent exercise diminishes it.

I would not wish to finish this subject without a word as to the reasons which seem to me to favour precipitation of uric acid in the joints so producing the symptoms of gout, as an explanation of this epiphénoménon should necessarily follow the foregoing remarks. This selection of the joints by uric acid, under certain conditions of the blood, seems to be due to lessened nutritive changes in them, and is not a mere coincidence; here, also, it seems to me, a lessened nutritive metabolism, or an atrophic condition, renders them vulnerable; in this process the conditions of their trophic nervous supply must play an important part. If the nervous system is to be especially invoked in gout, it is in the production of this vulnerability in these situations. The facts that have been accumulating since Charcot first called attention to the disease which bears his name, and to the intimate relation he proved to exist between the joint lesions and the spinal cord, through the influence of its trophic nerves, go to show that there is a defect in their nutritive metabolism in such lesions.

Nor are evidences wanting from the clinical side to show that in this precipitation of uric acid in the joints, the nervous system is upset, and its influence in preserving them impaired. We know an individual may go on forming or retaining uric acid in the blood for years, and no gout results until some depressing influence upsets his nervous equilibrium, and then gout becomes manifest. It

may be owing to a nervous shock, or to over-anxiety, or loss in business or reputation, or to an illness, or to an accident, but that it manifests itself under such circumstances we have abundant examples to prove. We all know how an accident to a joint will produce gouty pains in it afterwards, by interfering with the nutritive metabolism of its delicate structure, and it is often the first indication to us and to the patient that he is gouty—a fact which neither the patient nor the physician previously suspected.

In conclusion, the inference we must draw from this review of the subject is, that the gouty individual is a susceptible individual, whose general metabolism is easily upset, and that this vulnerability may arise from a disturbing influence in any of the great physiological systems I have enumerated—in the nervous, the digestive, and the circulatory systems especially—and to a lesser extent in the others I have mentioned, and no treatment can be successful which does not take note of this. We should earnestly inquire into and, by investigation, ascertain in which of these physiological systems the defect first arises—a matter of no small difficulty in many cases—as, owing to their inter-dependence on one another, defective metabolism in one may influence that in all the others (as I alluded to in the first part of my paper), and it may be almost impossible in the general confusion to single out the ringleader in this "vice of nutrition," and in doing so we should bear Ebstein's remark in mind, that the "gouty individual forms uric acid in perverse localities, even in bones." One fact we should bear in mind, that clearing the blood and system of uric acid by alkalies, or salicylates, leaves the principal part of our work undone—namely, the correcting of the faulty metabolism in whatever system it first arises by suitable remedies directed to them. What these remedies should be does not enter into the scope of this paper, but we know we can influence their metabolism for good by many means—medicinal, hygienic, and dietetic. We must also remember we are dealing with a complex problem, which is mainly a biological one, and that chemistry alone will not solve it for us, though it has done a great deal towards it. Whatever additional knowledge we are likely to gain—and much is still needed—must come from workers in the field of biological investigation, and it seems to me it is in

this direction that investigators should turn their attention for its complete solution.

A CLINICAL LECTURE ON STRICTURE OF THE URETHRA.

Delivered at the Central London Sick Asylum,
June 18, 1896,

By REGINALD HARRISON, F.R.C.S.Eng.,

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GENTLEMEN,—By the kindness of my friend Mr. Hopkins, I have for demonstration to-day one or two cases of stricture of the urethra, which I hope may prove instructive.

You are aware that urethral strictures may be divided into two classes: first, what I would speak of as inflammatory product strictures, which result from previous inflammations and most commonly from gonorrhœa. The second class is the variety which follows injury to the urethra, where the wall of the canal is, to some extent, involved in the scar resulting from the laceration. It is important to draw a distinction between inflammatory and traumatic cases, because we find in practice that the latter are more difficult to manage and often require more extreme measures than the former. I do not propose to discuss the pathology of strictures, because I wish to direct your attention particularly to its mechanical treatment and the variety of instruments we employ for this purpose.

By stricture, whether inflammatory or traumatic, we understand some narrowing of the urethra, which renders micturition more or less difficult. There can be no doubt that progressive strictures sometimes assume such a degree of contraction as to lead to destruction of the whole of the urinary apparatus behind them. We see, for instance, the bladder thickened in order to overcome the opposition to the passage of urine, the ureters dilated, and the kidneys eventually reduced to mere suppurating sacs.

Assuming that the patient complains of difficulty of micturition, frequency, and so forth, it becomes necessary that we should examine the urethra so as to ascertain whether any obstruction can be

detected. I usually proceed to examine the patient by placing him in the recumbent position. I know some surgeons prefer the erect posture, but I think the other is far more convenient, and the patient is, moreover, in a favourable attitude for muscular relaxation, which greatly assists the introduction of instruments, especially in inflammatory conditions.

With regard to anaesthetics, some strictures, as you are aware, are extremely sensitive, and directly the instrument touches the obstruction spasm is provoked. I frequently use a 10 per cent. solution of cocaine to prevent this. I apply this with a syringe specially made for the purpose, which is charged with a small quantity of the cocaine solution; this considerably aids one's efforts to introduce an instrument past the obstruction. Of course, with extremely sensitive persons, where this does not suffice, it is better to administer a general anaesthetic, at all events on the first occasion. Having placed the patient in a suitable position, I pass one of these elongated, tapering, soft bougies, which are known as "whips," and which I first described in the "Lancet" ten or twelve years ago. Where the stricture is not very tight the whip gradually finds its way into the bladder, and curls up inside that viscus; thus one is enabled to ascertain not only the position of the stricture, but also its size and the number of the bougie it will admit from the use of a single instrument. It is important to abstain from trying to force a stricture.

If the stricture is so tight that it will not even admit a whip, we should try to pass what is known as a filiform bougie. I show you a variety of them, some made of whalebone, others of gum elastic. With an extremely tight stricture, having passed a fine filiform bougie, I often tie it in with a little silk secured by a piece of plaster over the penis, and let the patient retain it as long as he can—48 hours if possible. The patient is not usually inconvenienced by this, but passes his water by the side of the bougie. At the end of the 48 hours one may often introduce a fairly large whip, and so effect a considerable dilatation in an easy way. There is a modification of the filiform instrument which is well known in America as Bang's filiform bougie. They are made of a very fine kind of whalebone, with a small bulb at the end. One of these will sometimes pass a tight

stricture when a flexible filiform will not, and thus rapid dilatation on the principle of the wedge may be readily effected. Another useful instrument much used in America for tight strictures is Gouley's tunnelled bougie, and it is employed in the following manner: A little oil is first injected into the urethra, and then a very fine whalebone filiform bougie is insinuated through the stricture; this having been done, a metal bougie is threaded on to the filiform instrument, which acts as a guide to the former on its way through the stricture—hence the term "tunnelled bougie." A catheter on the same principle can also be used where retention of urine exists. I do not think it is sufficiently known in this country. In ordinary cases I usually prefer the gum-elastic bulbous instruments.

But with all your care and the various devices I have just shown, you may sometimes find a stricture—particularly a traumatic one, where the cicatricial contraction is very great—which renders any entry into the bladder by means of a flexible bougie quite impossible. Then we have to resort to a metal instrument. After a considerable experience with all kinds of appliances, the metal bougies which I find of most service in these cases are Lister's, which were described by him when he was teaching in Edinburgh, and I have used them ever since. They have a bulb at the end, and are so graduated that each instrument between the point and the end of the curve may be said to represent three consecutive sizes.

Now a word as to gauging. To treat strictures successfully by dilatation, you must be uniform and systematic—that is, do not pass a No. 3 one day and a No. 7 the next, but go regularly up the scale. In order to avoid abrupt changes of size it is always well to employ a gauge, a specimen of which I show you. It is an angular slit in a piece of metal, and the gauges of three countries—England, France, and America—are indicated thereon, for unfortunately we are not uniform in this respect. I believe the English system is far too abrupt for many strictures, and for this reason I generally adopt the French scale.

The majority of strictures met with in practice, I feel sure, are best treated by some form of dilatation. The surgeon having effected a sufficient degree of dilatation, it is his duty to teach the patient how to use a suitable bougie, because if the

practice is not kept up, recurrence is certain to take place. The majority of relapses are traceable to the neglect of the patient in this respect, for as he finds the inconvenience diminishes so does he gradually cease to use his bougie.

On the other hand, there are strictures which are unsuitable for dilatation on account of their extreme rigidity and contractility, an attempt to do this sometimes bringing on rigors or fever. Fortunately cases which will not admit any instrument are comparatively rare, as during the last ten or fifteen years there has been a great improvement in all kinds of urethal instruments, and anæsthetics also help us greatly. There are several ways of treating strictures other than by dilatation, as, for instance, by various forms of urethroto my, but I do not propose discussing this phase of the subject to-day.

We will now look at the cases.

This male patient, aged 34, is the subject of stricture of four years duration, occurring after an attack of gonorrhœa dating thirteen years ago. The passing of a catheter is usually followed in this instance by a rigor and fever, resembling somewhat an attack of ague. This gives me an opportunity of speaking about the importance of sterilizing the urine in cases of stricture requiring the use of instruments. Many years ago it was pointed out—I think by the late Dr. Palmer, of Louisville, U.S.A.—that if certain drugs, such as quinine, boracic acid, salol, and others are administered to patients who are liable to rigors, those symptoms are often prevented, and I find this to be true. In the performance of internal urethroto my, I make a practice of giving the patient, for a week previously, boracic acid in doses of five or ten grains three times a day; this will generally render the urine fairly saturated with the salt. As to the pathology of urethral fever, I believe it is entirely bacterial, and that where means are taken to prevent the development of this germ life in the urine, the consequences I have mentioned do not take place so frequently.

This next patient represents a traumatic case. He received an injury to his scrotum some years ago through slipping over a cab wheel, and the part must have been extensively lacerated, so that the whole of the urethra is involved in a cicatrix, which extends, practically, from the root of the penis to the perinæum. This is a stricture of car-

tilaginous hardness, and thus corresponds with the description I have already given of this variety. We are told that his urine now passes in a full stream, a result following upon a perinæal section which was done on May 1st. The patient illustrates the good effects of this kind of treatment in traumatic strictures, but I fear relapse will take place if he does not continue to use a bougie.

Here we have another male with a cicatrical stricture, which he has had nearly seven years. He can pass the bougie himself, and when I ask him to try one of the whips I have been showing you to-day you hear how much he appreciates it in comparison with the one he has been in the habit of using for some years.

I should like you also to look at this prostatic case—a man 68 years of age. Here we have an example of the kind of obstruction met with in persons of advanced years. I will ask one or two of you to examine his prostate from the rectum, and you will at once recognise the nature of the obstruction.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM

BY

JONATHAN HUTCHINSON, LL.D., F.R.S.

Reported by J. T. CONNER, M.D.

Psoriasis limited to the Backs of the Hands.

The patient was a young woman æt. 25, sent by Mr. Edward Wallis, of King's College Hospital. On the backs of the hands were a number of pink-coloured patches, about the size of six-pences, slightly elevated, with a tendency to become circinate, and scarcely at all scaly. They were of three years' duration. She was in good health, and gave a history of a previous attack in the same place fifteen years ago, which lasted twelve months. No trace of eruption could be found elsewhere. Mr. Hutchinson believed the case to be one of psoriasis, which was peculiar in its limitation to the affected region. He mentioned the analogous cases of psoriasis confined to the finger-nails, shown at the demonstrations previously.

Jaundice treated by Mercury.

A Polish Jew, of middle age, whilst in good health, four months ago ate a dish of periwinkles. He felt distress at the pit of the stomach, and was sick. An eruption of urticaria followed. His health failed, so he was sent to Margate. There he "caught cold," and jaundice developed, a month after the beginning of his illness. This was accompanied with a good deal of pruritus, which, it was noteworthy, preceded the appearance of the jaundice.

He now showed the usual lemon-coloured tint of skin; but the tongue was clean, and the liver not enlarged.

Mr. Hutchinson mentioned another case in which indigestible food had produced the same effect. A man was sick from eating highly kept game. Urticaria and jaundice followed. But the case ran its course much more quickly—in three or four weeks.

The treatment he recommended was one grain of Hyd. c. Cret. twice a day. In the last case improvement at once occurred; though other treatment had been followed, without avail, for weeks. Physiologists now said that mercury had no action on the liver. Mr. Hutchinson begged to differ from them on the grounds of his clinical experience.

Incidentally he called attention to the peculiarities presented by the patient's finger-nails. They were well formed, hard, and very polished. This condition he always found associated with gout, and had not seen a better example of "gouty nails;" but they showed a further peculiarity, which he had found to be one of race—all the nails, even to those of the little fingers, showed well-marked lunulae. Most people had well-marked lunulae only on the thumb and adjacent two or three fingers. In Jews they were to be seen well marked on all the fingers.

Traumatic Dermatitis (Eczema?) of the Hands, infective to other parts.

The patient was a gas worker, æt. 45, sent by Mr. Hitchens. Five years ago, a spot of boiling tar fell on his left hand, which was followed by inflammation of the skin around the burn, lasting two months. Nine weeks ago the same accident occurred to the right hand. The whole hand, back and front, became inflamed and covered

with blisters the size of peas. The inflammation extended up the arm, producing smaller blisters at the wrists, and an erythematous condition as far as the elbow. Three weeks later the left hand and forearm became similarly affected, and the skin of the feet also inflamed.

The skin of the hands was red and swollen, roughened and scaly. The forearms were so also, but in less degree. The soles of the feet were desquamating in large flakes. The condition resembled a trade eczema, such as a potboy's. Mr. Hutchinson believed the tar was the exciting cause. The case was an example of the doctrine that all inflammation was contagious to the individual, but remained confined to the tissue primarily affected. In the first attack it had spread locally only; in the second to distant parts also. Some quite new observations had been lately made which exemplified this doctrine. A lady, who scalded her left forearm, was shown at the demonstrations a short time ago. Near the scald groups of herpetic-like vesicles formed, becoming gangrenous. He had seen a nurse in whom inflammation, with the formation of bullæ, spread up the arm from a whitlow. Dr. Galloway had brought forward a case in which wide-spread erythema had followed a burn.

Syphilitic Sycosis-Lupus of the Face and Neck.

A man æt. 38, sent by Mr. Hitchens, had been subject to this disease for eighteen months. No history of syphilis could be obtained. The moustache region was covered with crusted nodules and scars, which were healthy and clearly margined. The hair was scanty, as most of the follicles had been destroyed. At the nape of the neck the skin bearing the small hairs was in a similar condition. The case was considered syphilitic, and not lupus vulgaris, for the following reasons:—There was no "apple jelly" deposit. The crust was purulent, not dry and scaly. The ulceration would not be so irregular in lupus. The scar was perfectly healthy.

The treatment recommended was epilation, removal of the scab by washing, and the application of the acid nitrate of mercury; iodide of potassium to be taken internally. But the local treatment was much the more important, and would cure the disease by itself, because it was now a strictly local affection.

Adenoma Sebaceum accompanied by peculiar brown patches (like those of Xanthelasma).

The patient was a girl, *aet.* 10, brought by Mr. G. W. Sequeira. The most prominent feature was a bat's-wing arrangement of little red papules on the cheeks; most of which were discrete. On the right cheek, however, a considerable patch had been formed, apparently by coalescence, which was somewhat thickened. There was another such patch on the middle, and a third on the right side of the nose. Although many papules were florid, a considerable number were quite pale; nowhere was there any proof of sebaceous accumulation or of the orifice of a follicle. The upper lip showed only very small papules, and was indeed almost free. Between the prolabium of the lower lip and the chin were a considerable number, three of which were as large as small shots. The lesions were for the most part symmetrical. A very peculiar feature in the case was the presence of some light yellowish brown patches on the forehead and upper eyelids (like those of xanthelasma). They were irregular in shape, and varied in size from a pea to the tip of the little finger. On the left upper eyelid in the outer half were hypertrophied folds of skin which extended from the eyelashes to the eyebrow, and hanging down concealed the eyelashes and the outer canthus. The skin of these folds was soft and yellowish brown. There was the representation of precisely the same thing in an earlier stage on the opposite eyelid.

In the middle of the forehead, just over the bridge of the nose, was a pale patch of slight thickening of skin, but without discolouration.

Upon her body, back, and abdomen were a few scattered, slightly elevated spots, all quite pale and none larger than a split pea. As regards the history, it was stated that none were present at birth. At the age of a year and eight months she had a series of severe convulsions, lasting ten hours and a half. There were none before or since. Some weeks after the convulsions she had an attack of jaundice, and was very yellow for a month. After this the spots were noticed. It was stated that Mr. Langton excised a portion of the skin from the left upper eyelid two years ago.

Mr. Hutchinson said that the brown patches were an unique feature in the case. They were quite new to him. Adenoma sebaceum mostly

occurred in idiots or those subject to convulsions, but he believed that it was not produced by the disease, but by the bromide of potassium given for its relief. This drug, which could produce acne in the adult, might in the child have a more serious effect in the developing glands. He admitted that he could not always prove (as in this case) that it had been administered; but most of the cases had taken it for a long time. He also suggested that the jaundice might have been responsible for the colour of the yellowish-brown patches, having "stamped" them when it occurred.

Cheiro-Pompholyx.

The patient was a girl aged nine, in good health, sent by Mr. Waren Tay. For the last five years she had been attacked regularly every spring and autumn, first in the hands and then in the feet. She felt ill, but not to any extent at the onset. The present attack began three months ago as red pimples in the palms, which became transformed into vesicles, followed by exfoliation of epidermis. Now the palms showed large red patches from which the skin was peeling, the backs of the hands being quite exempt. They were unequally affected, the left was much the worse. A few scattered vesicles could be seen on the anterior surfaces of the forearms. The feet were but little affected, the soles showing only a few small red patches. The skin of the face and forehead was harsh, and on the latter some tiny vesicles could be seen. Mr. Hutchinson compared the case with one he had lately published ("Archives," vii, 165). A healthy girl, aged 14, was subject, for eight years, to an eruption of vesicles on the palms and sides of the fingers, whilst the backs of the hands were quite exempt. The intervals were six weeks to three months, during which the skin became perfectly sound. He pointed out that the essential features of cheiro-pompholyx (as shown in these cases) were a recurring vesicular or bullous eruption of the hands, and perhaps of the feet, which showed no tendency to travel up the forearms, or pass into an eczematous condition, and which subsided spontaneously. It was thus analogous to an ordinary catarrh. The subjects were liable to "catch cold" in their skin. In his experience, this liability was not accompanied by the same tendency in the mucous membranes of those subject to the disease. But other members of the family who were exempt might be liable to ordinary colds.

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A POST-GRADUATE LECTURE ON INTERNAL DERANGEMENT OF THE KNEE-JOINT AND LOOSE CARTILAGES IN JOINTS.

Delivered at the Central London Sick Asylum,
February 27, 1896.

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LADIES and GENTLEMEN,—In connection with the lecture, I thought it would be interesting if I showed you this patient, who has a loose body in the upper recess of the outer side of the knee-joint. The prominence it causes there can be seen, and is at once evident on examination.

The man is twenty-five, and has felt weakness in the right knee during the last three or four years.

In May, 1894, he had rheumatic fever, which affected his right knee, as well as other joints. The history of his present illness is that nine months ago he fell down a flight of stairs while carrying a sack of coals. He does not know definitely whether he struck the knee in his fall, but it became swollen and painful directly afterwards. The pain and swelling persisted for three weeks, but did not prevent him walking about. Since the accident he has always walked with a limp, and the knee feels weak. When walking, he feels at intervals a sudden pain in the right knee, which compels him to stop. He then has to begin walking cautiously, manipulation of the knee often becoming necessary to enable him to start. Sometimes the knee is definitely locked. These attacks are usually produced by any sudden movement, and the swelling which follows the attack varies in degree on different occasions. Three months after the accident the patient noticed a movable lump, and he thinks it has slightly increased in size. Its position varies, but it mostly

occupies the outer aspect of the joint, where it is always found when weight is put upon the limb. It disappears when the joint is flexed to a right angle. Sometimes it gets to the inner and lower side of the joint, and the joint cannot then be flexed until the body is pushed away to the outer side. At present there is some slight genu valgum, without effusion into the synovial cavity, or thickening of the membrane. On the outer side of the patella an almost circular hard disc can be felt, about the size of a halfpenny piece, freely movable within the joint, which is somewhat tender on manipulation. The joint movements are neither limited nor painful, but the patient continues to limp. That is the history of a loose cartilage in a joint, arising apparently in this case as the result of injury. On removal subsequently this body was found to consist of articular cartilage with distinct evidence of ulceration on its deep surface.

On the table are a series of interesting specimens. The first shows a small quadrilateral portion of cartilage $\frac{1}{2}$ by $1\frac{1}{2}$ inch, with a subjacent layer of well-formed bone. On three of its borders it is sharply cut vertically, and is striated, the smooth surface of the cartilage being continued to the thin edge of the bone. This piece of articular cartilage and bone was removed by Sir John Simon from a young man who had wrenches his knee in falling. He came to St. Thomas's with some synovitis, and as a result of the accident was found to have a loose body in the joint. As soon as the acute symptoms had subsided, three weeks after the injury, the body was removed.

The case is interesting as bearing on the genesis of these loose bodies, because it has been often denied that they can be formed by a traumatic detachment of the articular cartilage. P. Bruns considers it to be of the utmost rarity, and most German authors express a somewhat similar opinion. It is clear, however, from this instance, that such an accident is possible.

The next specimen is a flattened, oval, pedunculated body, consisting of pure cartilage, which was excised from the knee-joint. It measures

$\frac{3}{4}$ inch across by $\frac{1}{8}$ inch thick. The patient from whom the specimen was taken had fallen from a horse, injuring his knee. Eighteen months after the fall symptoms appeared indicating what was believed to be a displacement of the external semilunar cartilage. The lump could be felt behind the joint, on its outer side, and a vertical incision was made, under the impression that it was a displaced cartilage. The semilunar cartilage was found to be normal, but on introducing the finger into the joint, this movable pedunculated body was brought to the surface and removed. The patient recovered slowly, in consequence of suppuration having taken place in the joint. He had afterwards a useful, though somewhat stiff joint. Here again is a point of interest, namely, that these bodies should, if possible, be removed without manipulation in the interior of the joint, as this necessarily increases the risk of suppuration.

The next specimen is a flattened oval body, consisting of a pedunculated portion of cartilage $\frac{3}{4}$ inch in diameter. In this case also there was a distinct history of injury, the patient having struck his knee against a stone buttress while playing at football. Acute synovitis followed, and symptoms pointing to a loose body occurred some months later.

These two examples of flattened cartilaginous bodies, with a small amount of well-formed bone in each, were found, one in each of the two knee-joints of a boy aged 15. They were removed in 1885. The boy had noticed nothing wrong with the knees until seven weeks previously, and then experienced, without known cause, acute pain, which prevented him walking. After the removal of the bodies the patient made a perfect recovery. In this case it is possible there may have been a traumatic cause, the injury being forgotten or overlooked by the boy.

This specimen presents another instance of loose body composed of cartilage and bone, while the next is the articular end of the femur, belonging to the joint in which the loose body lay. By the unfortunate death of the patient an opportunity was afforded of ascertaining that the loose body actually corresponded to a gap in the articular surface of the condyle, from which this body has clearly come. There is also evidence of healing or cicatricial change in the part of the joint surface from which the body was detached.

Next we have an example from a case in which a large number of bodies were removed from the knee-joint after death, and I need only say that some of them are composed of cancellated bone, and others are cartilage. The number sometimes amounts to hundreds, and in one case Mr. Berry recorded 1047. ("Brit. Med. Journal," 1894.)

This preparation is of considerable interest; it shows the lower ends of both femora, removed after death. You will notice a circumscribed partially detached portion on each condylar surface, which, when completely separated, would have formed a loose body in each of the two joints, separation being, as you see, in actual progress.

The next preparation shows an old blood-clot, which has apparently formed in the synovial border, and which might possibly give rise to a loose body. It is a well-known mode of origin of some of these bodies.

The last specimen I have to show you belongs to another series, namely, a case in which a portion of the internal semilunar cartilage was torn away from its attachments, and subsequently excised from the knee-joint.

The cases of loose body which have been admitted during the last few years into St. Thomas's Hospital and operated on there have been eighteen in number, including two cases of detached semilunar cartilage; and it is of interest to note that of these seventeen were males, and only one was a female. The ages of the male patients varied between 15 and 44, the age of the female being 53. There was a distinct history of accident in five, but the specimens preserved in the museum which I have shown to you indicate a larger proportion of accidental causes. As to the nature of the bodies removed:—

Those consisting of pure articular cartilage	4
" " articular cartilage and bone 4	
" " cartilaginous body arising from fringe	2
" " fibrous body from fringe	3
" " tuberculous mass forming in a synovial fringe 1	
" " osteophytic growth	2
" " detached semilunar cartilage 2	
	18

Loose cartilages in the joints are formed in different ways, and I have pointed out the variety of their composition. In addition there are the melon-shaped bodies, of which I have not a specimen to show you. These are fibrous concretions, more frequently met with in the bursæ and tendon sheaths than in the joints, and they are frequently associated with tuberculous disease.

As regards the treatment of these cases, it is important to remember that some of the bodies are pedunculated, while others are free. The specimens I have shown demonstrate how variable they may be in form—oval or circular plates, concavo-convex plates, biconcave, biconvex; or they may be rounded bodies like a boy's marble, or as large even as a walnut. In many instances they are single, or at most but two in number; while in others there may be six, a dozen, twenty; and hundreds of these loose bodies have been occasionally removed from a single joint.

With regard to their location, nearly all the instances I have quoted were cases in which the knee-joint was involved. This joint seems to have a preponderating share of these loose substances. Ashhurst, a very careful statistician, calculates that as many as 85 per cent. of the total number of loose bodies found in joints are met with in the knee alone.

Concerning their origin, they may occur as the result of injury, though, that, it has been generally contended, is somewhat rare; I mean the direct chipping off of the bone surface. Among the specimens I have shown you, one very well illustrates this mode of origin. By many authorities it has been altogether denied that such a course is possible, and it is difficult to conceive the mechanical possibility of it; still it seems fairly certain that cases do occur in this way. A frequent mode of origin of these bodies is some antecedent inflammatory change in the joint, often dependent on traumatism. Perhaps one of the common ways in which this takes place is for some part of the bone ends to be severely contused; for instance, while the individual is in the kneeling position, or the cartilaginous surface of the femur is exposed to a direct impact of the force, and this, when violent, may bruise the cartilage and the subjacent bone. As a result of the contusion, inflammatory changes occur, which finally terminate in the separation of a portion of the articular sur-

face, with a certain thickness of subjacent bone, by what is known as "quiet necrosis," without the formation of pus. It has been called *osteochondritis dissecans* by König, and is believed by some to arise independently of an injury. That this is certainly the origin of some of these bodies is proved by examination, upon which it is evident that one surface is covered with normal articular cartilage; while underneath, and closely attached to this articular cartilage, is a layer of cancellous bone, such as would be found in the articular extremity of the femur. Not only has it been proved in this way, as in the case of the man showed you at the beginning of this lecture, but it has been actually verified by examining the interior of the joint in fatal cases and finding there was a gap corresponding to the portion of bone and cartilage which was found loose, and in several cases of operation the gap whence the body was extruded has been plainly visible. Sir John Simon's case is one of traumatic detachment of a portion of the articular surface, which was removed only three weeks after the injury, and was then found to be true cartilage and bone. In another case, mechanically less difficult to understand, Mr. Bruce Clarke found a portion of detached bone and cartilage in the joint, which had come from the articular margin of the patella.

Max Schüller has made an interesting contribution to the question of the traumatic origin of these bodies, in a paper published in the *Aerztliche Sachverständiger Zeitung*, February, 1896. He collected from various Continental, English, and American sources 143 cases, which were operated upon during the years from 1883 to 1893.

Of these he found 85 had a distinctly traumatic origin, 39 were due to pathological causes, and in 19 the cause was unknown. In the first category the symptoms of loose body appeared in many instances in a few days or week after the injury, and portions of but little altered joint surface were removed.

Of the 85 cases 78 affected the knee, five the elbow, and two the wrist.

On analysing the causes, he found two main ones, either forcible movement, that is movement beyond the normal joint range, and twisting movements, or external violence applied to the articulation, and in some instances both causes were in operation. He points out that any very

excessive force is not required to cause the detachment of a portion of the articular cartilage and the subjacent bone, which may be either torn or split off or squeezed out, and may sometimes remain attached by shreds of capsule or ligament, or else be completely free.

He regards a traumatic origin as being much more frequent than is generally supposed, and remarks that although osteo-arthritis and tubercle are very common affections, loose bodies in connection with them are comparatively rare.

In 1883 Virchow showed at the Berlin Medical Society a piece of cartilage and bone, which Karpinski had excised from the knee of a soldier very soon after a jump from a considerable height. It was a portion of the condylar surface $2 \times 3\frac{1}{2}$ cm. in diameter. Riedel removed one from the knee four weeks after a severe blow, similar in size, which had been separated from the internal condyle of the femur; and he also excised, 17 days after the injury, a somewhat smaller piece of articular cartilage and bone from the knee of a girl of 15 who had sustained a violent twist of the limb, the body being bent at the same time to the affected side.

Robinson removed a body $1\frac{1}{8} \times \frac{5}{8}$ in diameter and $\frac{1}{4}$ in. thick six days after a fall of a heavy weight on the knee in a boy of 16, and Hingston, of Montreal, removed a broken-off piece of joint surface $1\frac{1}{2}$ in. in diameter seven days after the injury.

Schüller gives an interesting case of his own of a man of 30 who fell on the bent knee. There was much effusion at the time, and four weeks after, when he became able to get about, symptoms of loose body were manifest. Six months after the accident Schüller removed a piece of cartilage and bone $2\cdot5 \times 2$ cm. in diameter, and during the operation was able to see the depression in the articular surface precisely corresponding to the size and form of the loose body removed. He also records a similar case where a portion of the capitellum of the humerus had been separated after a fall, and here too the gap in the bone whence the fragment was detached was visible during the operation.

Then we have cases in which an accidentally intruded foreign body, such as a needle, has formed the nucleus of a loose body; while others may be the result of the hardening of clots of

blood. Hunter believed that this was the common mode of production of these bodies, but I think that is certainly not the case. No doubt haemorrhages in connection with joints do often give rise to these bodies, but it is mostly by an extravasation of blood into one of the synovial fringes; the blood may be transformed into a hard body or become organised; afterwards it becomes more or less pedunculated, and may either remain so or become detached. Such bodies have been removed from individuals who have sustained injury at football and other games.

Besides extravasation of blood into the synovial fringes, there may be proliferation of the cartilage cells normally found there; these may take on growth and small nodules form, which gradually become larger. They may be either fibrous, cartilaginous, or bony, and as the result of their weight the fringe becomes elongated, the nodules become pedunculated, the peduncle may gradually elongate, and finally the body may become detached, or else it remains attached.

In cases of osteo-arthritis, the outgrowing lips and bony prominences at the joint margins may sometimes form loose bodies.

The diagnosis, in such a case as that presented to-day, is made with extreme facility; the possibility of mistaking the nature of the lesion is very small, and so it is in many instances. But you can understand that in some the body is not so easily brought to the surface. It has a habit of lurking in the recesses of the articulation, a habit which accounts for the German name *Gelenkmaus*, and the surgeon may find great difficulty in discovering it. Very often he has to secure the assistance of the patient, who, by experience, knows by what kind of movement he can push the body out of the interior of the joint. Another difficulty is that when you first see the patient you may have to deal with a swollen joint, when it will be necessary to wait until the synovial effusion subsides before you can make a satisfactory examination of the case. The history, of course, may point strongly to the nature of the affection. The patient has usually experienced a very sudden pain, which is often described as agonizing, and with its onset the joint becomes immovable, being locked in a semi-flexed position, so that the patient can neither extend nor bend it. If he has nothing to support himself by, he probably falls to

the ground, and is seized with sickness and faintness, which may last a few seconds or a considerable time. Then, by some movement of the joint, he may be able to release it from its locked condition and proceed on his way. The pain produced by the body, sometimes of intense severity, is probably caused by the movable cartilage or body getting in between the joint surfaces, and, acting as a sort of wedge there, occasions a great deal of pressure on the sensitive bone beneath; and it also, by its fulcrum-like action, violently stretches the ligamentous structures. Locking of the joint always occurs in the semi-flexed position, and it may last a few seconds, minutes, or an hour or two; an attack of synovitis always follows, necessitating treatment for some days. A further point in the history of these cases is that the patient has similar attacks at intervals without warning, and each time has to lie up in bed. As these attacks recur, the joint becomes more and more deteriorated, the synovial membrane is apt to become thickened, the effusion disappears more and more slowly each time, and may eventually refuse to subside. This leads one to remark that in cases of chronic synovial effusion into the knee, for which there is no other or obvious cause, it may be well to remember that the condition is sometimes caused by the presence of loose bodies, and emphasises the necessity for their prompt removal so soon as their existence is definitely ascertained.

It has been pointed out that this locking of the joint may, in some cases, occasion loss of life. For instance, an individual swimming in deep water may, in kicking out, get such a mass of cartilage between the joint surfaces, and the locking, coupled with the extreme pain which it occasions, may prevent him keeping above water. As I have said, these bodies are most often met with on the outer surface of the joint, between the patella and the condyle. They are less frequently found projecting on the inner side. When the body is found, it should be fixed on the lateral upper aspect of the joint by strapping or bandaging; or, if you are about to excise it, it should be first transfixed.

The treatment of these cases has become increasingly effective since the introduction of antiseptic methods. Formerly there was, very properly, such a dread of opening the synovial cavity, in

consequence of the frequently disastrous results which ensued in the absence of antiseptic precautions, that these bodies were removed by a subcutaneous method of operation, and very good results were thus obtained. A long, strong tenotome was introduced two inches above the joint, the body having been previously fixed, either by the fingers or preferably by transfixion with a needle, or by strapping in the upper, outer recess of the joint, and then the capsule was freely incised over the body, which could then be extruded from the joint. Mr. Square, of Plymouth, many years ago introduced a notable improvement in the subcutaneous method, which consists in making a bed outside the capsule for the body to lie in after it was pushed out from the joint. Previously, the body would often return again to its former position, and thus neutralize the effect of the operation. There is another cause of failure of the subcutaneous method easily understood, namely, that the body may be pedunculated (and in a certain number of cases is thus attached by its pedicle). The subcutaneous method fails in such cases because the body cannot be pushed out or will slip back again after being extruded unless its pedicle can be ruptured. I remember dealing with a case of this kind. Having made the incision through the capsule and a bed for the body to lie in, it refused to go into its new position, and only by being able, after exerting considerable force with my thumbs, to rupture the pedicle, could I bring the operation to a successful issue. In many other cases the pedicle is too strong to be ruptured in this way, and the subcutaneous method will fail. A former plan of transfixion with a pin, and thus endeavouring to set up inflammatory adhesions, need hardly be discussed. In the absence of antiseptic precautions, it is dangerous, and is also very likely to be ineffective, because the adhesions are, at best, only trivial, and the movements of the joint are likely to break them down, when the body would resume its movable condition. Mr. Barwell's statistics of the indirect method showed a failure of twenty-five per cent. of all the cases, and of course the method mostly failed in cases of pedunculated bodies. The practice has now been practically abandoned in favour of the direct method of operation, which consists in moving the body to a convenient place in the joint, pre-

ferably to the upper outer aspect, where the tissues are comparatively thin, and, having transfixed it there, to cut down and remove it. On that point there is only one warning necessary ; you must be sure, before operating, that the body is so fixed that it will not escape, otherwise it will very easily slip out of reach, the fingers alone, unless the body be bulky, are insufficient for the purpose. I advise you, therefore, if you have such a body to deal with, to first pin it firmly down to the bone before you make an incision. I have known more than one case in which the surgeon, trusting to his fingers or to those of his assistant for the needful fixation, has found, after making his incision, that the body had escaped from control and was no longer within reach. The operator had then either to abandon the operation—a sufficiently unfortunate proceeding—or to explore within the joint in various ways, to get the body to reappear. In one such case I can recall, suppuration ensued, a condition which may terminate in an ankylosed limb or worse, and I have known another case in which such a state of things cost the patient his life, infective changes leading to pyæmia taking place. But these cases of loose bodies, if operated upon in the way I have described, with proper precautions are exceedingly satisfactory, and there is very little, if any, risk to life or to the function of the limb. There is no bleeding into the joint, therefore no need to wash it out. After the operation the wound in the capsule should always be closed by buried sutures ; the limb is subsequently put up in some form of splint to immobilize the joint for ten days or a fortnight, and when the wound is healed, passive motions may be begun, active movements following later. Recovery is usually rapid and complete.

In illustration of the second part of the lecture, I have only the one specimen already shown to you. It is a portion of internal semilunar cartilage which was removed from the knee-joint. It had been torn from its attachment by injury in a patient aged 39. Three weeks previously he had slipped, his foot gave way, and he fell upon his right knee. He was unable to straighten the limb. During repeated examinations the internal semilunar cartilage could occasionally be found displaced, and flexion caused pain. The joint was opened by a transverse incision over the cartilage,

which was found attached at its anterior and posterior extremities, but separated around the margin, and curled up in the inter-condylar notch. As much of it as could be conveniently cut away was removed, and the patient made a complete recovery.

I recently had occasion to operate on a young gentleman who seemed to have displaced his internal semilunar cartilage ; he himself termed it a "football knee." At the time he experienced sudden severe pain, during an effort at football, followed by flexion and inability to extend the knee. He states he twisted the limb outwards while in a partially flexed position of the joint. This was followed by effusion in the joint and inability to use the limb. When he came to me he had had twelve painful attacks of this kind, apparently excited by any unusual movement of the joint ; each one was followed by synovitis of similar character. At the time of the attack the knee was not exactly locked, but was semi-flexed, incapable of being extended ; and any attempt to extend caused pain. Synovitis appeared immediately after the attack, which was sometimes brought on by a most trifling twist of the limb. This caused him to lie up ; in short, he came to me in a perfectly crippled condition. He was, he said, never sure of himself, and quite unable to follow his profession of a soldier, and with the best part of his life before him, was naturally desirous for something to be done. I examined him carefully, and beyond a fulness on the inner side of the joint between the patella and the margin of the condyle, I was unable to make out anything wrong. But the history pointed to a condition of displaced internal cartilage ; and, as the patient was in such a helpless condition, I decided to perform an exploratory operation. I believed I would find a loose or displaced semilunar cartilage. Accordingly I cut down upon it, and found it quite loose ; and its slipping in and out had evidently caused the symptoms I have described. I excised nearly the whole of the cartilage, and the patient recovered excellently. The only difficulty in the operation was that the numerous attacks of synovitis had occasioned so much local congestion that every stroke of the knife was followed by general bleeding, from no particular point which one could seize, and this haemorrhage proved exceedingly

troublesome. When I separated the semilunar cartilage from its anterior attachment, the cut surface began to bleed freely, and I had to plug the joint with iodoform gauze for a time to exert pressure upon the bleeding surface. In consequence of this unfortunate complication, blood found its way into the interior of the joint, and I thought it safer to irrigate the interior with a solution of perchloride of mercury, 1 part in 4000 parts of sterilized water. A drainage-tube was also kept in for forty-eight hours, which gave exit to a copious discharge of serous synovia, blood-stained at first.

A fortnight after the operation I allowed him to bend the joint a little, and now, five weeks after operation, he can bend it to more than a right angle without pain or inconvenience. There were some slight adhesions, which I broke down under an anaesthetic, the breaking down consisting in supporting the thigh and letting the leg fall by its own weight, during which the adhesions could be heard separating. No reaction occurred afterwards, and I do not doubt that everything will now be right, and that he will recover a good and useful limb, and he certainly cannot suffer from any looseness of the semilunar cartilage on the inner side of the joint. I have since seen the patient, and find the function of the joint in all ways perfect.

This condition was first described by Hey in his "Practical Observations on Surgery," 1803; he called it "internal derangement of the knee-joint—a very good term."

With reference to the causation of the displacement, it may be of interest if I remind you of one or two anatomical points in connection with these cartilages. The internal and external semilunar cartilages are crescentic bodies attached to the tuberosities of the tibia by the coronary ligaments, and they are also attached firmly by their anterior and posterior extremities, which are thin and pointed, to depressions in the bone in front and behind the spine of the tibia. These cornua are also connected together by transverse bands, sometimes of considerable strength, at others very thin, and occasionally altogether absent. The external border is thick, and the internal border quite thin and free. They serve to deepen the articular surface of the tibia, and afford a freer means of play of the femur upon the tibia. The internal cartilage is elongated from before back-

wards. The external is more circular in outline, much less firmly connected with the tibia than the internal, and it certainly possesses a wider range of movement, partly because the external coronary ligament is pierced by the popliteus tendon. In a position of mid-flexion, a considerable amount of rotation movement is possible at the knee, and during extension there is an associated slight rotation outward of the leg, while during the movement of flexion there is a slight amount of inward rotation.

The external cartilage is, as before stated, much the more movable, and might be supposed to be the one more frequently displaced. But it is the internal semilunar cartilage which is much the more frequently displaced, probably four or five times as often as the external. The accident seems to occur most frequently by a rotation movement of the leg outwards, or, if the leg be fixed, rotation of the thigh inwards, which would amount to the same thing. These movements seem to exert a pressure upon the cartilage, which, if strained to breaking limit, will cause tearing of the coronary or transverse ligaments and separate the attachment of the semilunar cartilage in front or behind the spine to a greater or lesser degree; or the convex border may be separated, the extremities remaining attached. The anterior extremity is perhaps most frequently torn away, and folds backwards on itself. In an operation done by my colleague, Mr. Croft, he found the cartilage fixed at its anterior and posterior extremities, but the coronary border between was detached, and the cartilage displaced into the intercondyloid notch; and the cartilage was also split. The cartilage may be separated, but less frequently at the posterior attachment; and the external semilunar may be torn off and split in a precisely similar fashion to the internal, but, as I have said, this is comparatively rare.

Again, as in the case I have quoted, without any actual laceration being apparent, the cartilage may be so loosened as to slip in and out from between the tibia and femur. The cartilage may be split either longitudinally or transversely, and the detached portion project into the joint cavity.

The usual cause of the displacement is a forcible, or in some cases only a slight, twist of the leg on the thigh in the partially flexed position of the knee. When the leg is twisted out, or the leg

being fixed the thigh is twisted in, the strain is brought to bear upon the internal semilunar cartilage, and when it reaches the breaking point, the ligamentous structures controlling the position and mobility of the cartilage give way in one of the manners already referred to, and the cartilage becomes displaced. The converse movement (of the leg inwards or of the thigh outwards) would direct the force on to the external cartilage, causing a similar displacement of it.

The diagnosis of these cases may be easy, or very difficult. Of course it is needful to exclude the presence of loose bodies in the joint, which may give rise to similar symptoms. If you meet with a case in which the accident is of recent occurrence, you must rely upon the history and nature of the injury, which is followed by pain and by fixation of the joint in the flexed position, with complete inability to extend it. Although the displacement may be produced by no great amount of force, there will be the history of rotation outwards or inwards, and in some cases you will find distinct fulness opposite the line of the articulation or at the side of the patella, when the cartilage is curled up in the intercondyloid notch. In other instances you may be able to feel a distinct gap at the line of the articulation. Then of course extension and flexion of the joint will alter this fulness or gap, either removing the gap or increasing the fulness, or transferring it to another part. The accident is followed by synovial effusion, which while present will mask any irregularity; and it is necessary to await its absorption. In cases where the accident has occurred two or three times, it will be found that the displacement and subsequent effusion takes place more readily each time, until finally the individual loses the sense of security in the limb, as any slight twist may bring on the horrible pain and fixation of the joint. He is not able to run, or if he does it is with a stiff knee and the foot turned out, and with the constant sense of imminent risk of recurrence; nor can he participate in any games or exercises.

Now in the treatment of these cases if the surgeon may entertain a good hope that if he has to deal with a recently displaced cartilage, careful treatment over a prolonged period will give time for fresh adhesions to form which sufficiently fasten the displaced body in its normal situation. Of course the first thing to do is to reduce the

displacement. This may usually be effected by fully flexing the limb and subsequently extending it, with manipulation by the fingers in the opposite direction to that in which the cartilage has been displaced. One knows the reduction will have taken place when the normal movements of the joint are restored; and then prolonged immobilization, followed by cautious use of the limb with a special support, such as can be obtained from a good instrument maker, as a hinge splint for the knee with a pad over the displaced cartilage. These splints are excellent, as they enable the patient to get about and prevent the twisting which is so liable to cause a renewed displacement. With the early application of such an appliance, the patient has a very good chance of recovery. If, on the other hand, the accident has happened several times and this treatment has not been adopted, it becomes a question whether, after failure of other treatment, an operative proceeding should not be resorted to. Opening the knee-joint is a very serious matter, and more serious for this affection than for loose bodies, because in the latter case the body can be pressed into a convenient position, cut down upon, and removed at once, the opening being instantly closed. But for detached semilunar cartilage one has to perform certain manipulations within the joint, consisting either in the replacement of the body and its retention by means of sutures, or, in cases where this is not suitable, excision of it altogether.

In 1889 Mr. H. Allingham published a valuable monograph on this subject, giving an account of the cases which had been operated upon up to that time, including several of his own, and he strongly advises operation in cases which are not open to other forms of treatment. He suggests—which I think is important—that instead of the transverse or oblique incision (I am still speaking of the internal cartilage) a vertical incision be made between the patella and internal lateral ligament. My own experience of the vertical incision is that it affords adequate access to the interior of the joint and to the cartilage. Probably only in the comparatively young, in whom a sound limb is a necessity, will any operative procedure be contemplated, though the position and avocation, and the patient's willingness or otherwise to wear a support, will influence the decision. But the surgeon must

be sure he can operate aseptically, and must not forget that the possibility of a stiff joint cannot be excluded, even when all possible precautions have been taken.

The incision as recommended by Mr. Allingham should be two and a half or three inches in length in the long axis of the limb, and three-quarters of an inch or so internal to the patella, its centre corresponding to the line of the joint. If possible, the limb should be maintained throughout in one position to prevent blood getting into the cavity. The structures from the skin to the synovial membrane should be divided seriatim, until the division of the capsule exposes the synovial membrane clearly in the bottom of the wound. All the superficial structures should be divided throughout the whole length of the wound, and only after all bleeding points have been secured should the synovial membrane be opened ; this is easily done by pinching it up, slitting it, and enlarging the opening by means of a knife or scissors. The cartilage will now be fully exposed, and can be dealt with according to the conditions met with.

In regard to the position of the limb, there is more room in the flexed position, though either that or the extended one may be adopted. The condition of the cartilage will determine whether it should be excised or sutured. If suturing be chosen, as in cases where the cartilage is simply too movable, the method introduced in 1883 by Professor Annandale of Edinburgh, should be employed. Two (or more if required) sutures of silk are passed through the fibrous investment covering the tuberosity of the tibia, then through the cartilage, and tied. This will serve to fix it. Silk should be preferred to gut, as it is more reliable. There is, however, still the risk that the movements of the joint may loosen the sutured cartilage, so that the result is not so certain as when excision is performed. If the cartilage be fully separated from either its anterior or posterior attachments, the coronary ligament torn through, and the cartilage curled up, it is better, I think, on the whole, to excise it. There is, no doubt, much to be said for the physiological advantage of the other form of operation ; but it has been found by experience that the patient walks exceedingly well notwithstanding the removal of the cartilage, whereas in suturing there is, as I have said, the risk that the sutures will not hold.

When the cartilage is split or torn, the torn part should be excised.

With regard to subsequent treatment, if any blood have entered the cavity of the articulation, the joint should, of necessity, be washed out, a weak antiseptic solution of bichloride of mercury, 1 in 4000 of sterilized water, being probably the best. The washing should be continued until the fluid comes out quite clear. If there be no entry of blood into the cavity, the washing out should be dispensed with, for then there will be less risk of subsequent adhesions. Of course, where there is any doubt, irrigation should be performed, so as to guard the patient against all septic risks. After irrigation, or if the joint has been in any way irritated, a drainage-tube must be left in for twenty-four to forty-eight hours, or the joint will become distended with fluid.

The wound should be closed with great care, the synovial membrane, the capsule, and the external structures being sutured separately. After the dressing has been applied, the limb must be immobilized. For an operation such as this I find a modification of Thomas's hip splint, hinged at the hip, is an admirable apparatus. It enables the patient to sit up in bed the day after the operation, without any chance of disturbing the parts about the knee, and anyone who has been forced to lie in one position for a long period, especially if that position be on the back, will appreciate what a great relief it is to be able to change one's position.

As soon as the external wound is soundly closed ---two weeks or less after operation---slight passive movements may be cautiously begun, followed, as soon after as possible, by active movements. If any adhesions occur and persist, they may be very gently broken down under chloroform. If all goes well, and no mishap happens during the operation, a satisfactory result will be obtained.

Whooping-Cough.—Dr. Fisher concludes from the results he has obtained in the quinine treatment of pertussis that it is the best remedy for whooping-cough at present known for the following reasons : (1) It diminishes the number of attacks essentially in five days at the latest. (2) It reduces even the most vehement whooping-cough to a mild bronchitis in from twelve to fifteen days (3) It influences most favourably a possibly existing broncho-pneumonia.—(*Medical Record.*)

An Analysis of 23 cases of so-called CONGENITAL TUMOUR OF THE STERNO-MASTOID MUSCLE.

With Remarks
on the Treatment of Congenital Torticollis.

BY
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DURING the years 1892, '93, '94, and part of '95 I made notes of all the cases of so-called congenital tumour of the sterno-mastoid muscle which came under my notice at the North-Eastern Hospital for Children, with the intention of following them up at a later period in order to determine their relationship with torticollis. I have not succeeded in tracing all the cases, but, as they are more numerous than any series hitherto reported by a single observer, a short analysis of them may be of interest.

During the three and a half years mentioned 23 cases came under my notice. They were all brought to the hospital on account of a lump in the neck, which had been observed by the parents or the nurses. That as many as 23 cases should have come under the notice of a single surgeon during three and a half years is proof of the great frequency with which the condition occurs. All the cases were extremely well marked.

Fifteen cases occurred in males, and eight in females. The difference between the numbers in the two sexes is doubtless related to the larger average size of the head in the male sex.

It is generally stated that the commonest predisposing cause of the condition is breech presentation. The explanation which has been offered of this supposed greater frequency of the affection in breech presentations is that when the trunk is born and birth of the head is delayed, traction is made upon the legs, and that in that way one or other sterno-mastoid muscle is severely stretched and lacerated.

In my series the breech presented in 2 cases, the feet in 2, and 2 were cross-births. In both the breech cases the head is stated to have been born easily; forceps were not required; the mother

was unable to say whether the legs had been pulled upon or not. In the two footling cases there was difficulty in delivery of the head. The feet were pulled upon in one, and in the other it was necessary to apply the forceps to the head. It was not known whether traction had been made on the feet before the forceps were applied. In both cases of cross-birth the child was turned and delivered feet first, and in one of them the clavicle was broken.

My cases show conclusively that, however predisposing the after-coming head may be, a considerable number of cases occur in head presentations. In my series of 23 cases no less than 17 occurred in head presentations. The relative proportion of cephalic presentations (head, face, and brow presentations) to pelvic presentation (breech and foot presentations and cross-births) is 96 to 4. In this series the proportion is 17 to 6. Consequently, although the large majority of my cases occurred in head presentations, it is not possible to deny the special proneness of the accident to occur in breech presentations. At the same time the special causative relation of breech presentations loses much clinical importance in the face of the fact that in 23 cases the presentation had been cephalic in no less than 17. But with regard to these latter cases, the causative influence of traction or injury with forceps seems established, for the forceps had been employed in nearly half the cases, viz. in eight, which is far above the average of forceps cases to natural deliveries. Quisling ("Archiv für Kinderheilkunde," Bd. xii., Hft. v. and vi.) states that in pelvic presentations it is chiefly the muscle of the side which lies most anteriorly which is injured, whilst in head presentations it is the muscle of the side which lies most posteriorly. These are points which cannot be determined unless the position of the head is known with certainty in a large number of cases. It is not fair to assume that the majority of the cases occurred in first positions just because the first position is the most frequent. But if for the sake of argument we assume that the majority of the cases would occur in the common position, my cases do not bear out Quisling's dictum, which appears to have been founded on a study of nine cases, of which only five were observed by himself. In the first pelvic position the left muscle would be the anterior one. In five out of my six cases

in which the head was born last, the affected side was noted, and was invariably the right. In the first head position the posterior muscle would be the left. In sixteen out of seventeen of my cases of vertex presentation, the side affected is noted. Forceps were used in eight cases, and the injured muscle was in four cases the right, and in four the left. The child was born without any assistance or interference in eight cases, and the injured muscle was in four cases the right, and in four the left.

The occasional connection between torticollis and congenital sterno-mastoid tumour is certainly sufficiently well established to warrant the medical attendant in warning the parents about it, in order that, if it should occur, it may be noticed early, and that suitable treatment may be adopted before the deformity is marked and before the development of the affected side of the head is interfered with.

In the early stages, and in the slighter degrees of torticollis, treatment does not of necessity involve operation. Much may be done by frictions, manipulations, and voluntary exercises. The head should be held in the best position attainable several times a day, and the child, if old enough to assist in its treatment, should be encouraged to exercise in front of a looking-glass. She should hold a weight in the hand of the affected side, and then try to incline her head to the opposite side, and turn her chin to the same side.

In more marked cases, and in every case where there is asymmetry of the two sides of the face, the sterno-mastoid should be divided. Opinions differ as to whether the operation should be performed subcutaneously or through an open wound. I am strongly in favour of the latter method. I have divided the sternal and clavicular origins of the muscle subcutaneously several times without any mishap; but I have never dared to divide by this method the bands of cervical fascia, which at once come into prominence after the sterno-mastoid is divided, and after doing the open operation the risks of the subcutaneous operation have been strikingly apparent. The anterior, external and internal jugular veins and the phrenic nerve have been exposed, and might easily have been wounded. In these days of aseptic surgery, the only argument in favour of subcutaneous tenotomy is the small and almost imperceptible scar which results from

it; whilst in favour of the open operation there is the possibility of dividing the bands of cervical fascia without injuring the important structures above mentioned. There are at least three cases on record in which large veins in the neck have been wounded in the subcutaneous operation. In a slight case where the sternal head alone required division the subcutaneous operation would suffice, and would be free from risk. Division of the muscle midway between its origin and insertion has been recommended; I adopted that method in one case, but did not detect any special advantages in it, Mikulicz ("Centralblatt für Chirurgie," 1895, No. 1) advocates the removal of the whole of the contracted muscle except the portion of it which is traversed by the spinal accessory nerve. Surely the disfigurement caused by the long scar and the absence of the muscle would be worse than the deformity left after a less radical operation.

The open operation may be conveniently done through a vertical incision an inch to an inch and a half in length, situated over the interval between the two heads. The wound may be drawn inwards over the sternal head, which may be gradually divided from without in. The wound may now be drawn outwards over the clavicular head, which may also be divided from without in by cutting gradually through its fibres. The anterior jugular vein, which lies beneath, may be easily avoided. There will now be a considerable gap in which the cervical fascia is exposed, and on inclining the head to the opposite side strong resisting bands of fascia will become evident. These may be cautiously divided. When this is done the internal jugular vein, the scalenus anticus muscle, and very probably the phrenic nerve will be exposed. After every bleeding point has been secured the wound may be completely closed. The child should be made to lie as much as possible on the affected side, with its head resting on a large and rather fine pillow, with the object of stretching the muscles and other tissues which have undergone adaptative shortening. At the end of a week, by which time a wool and collodion dressing will usually suffice, the effect of this position may be supplemented by manipulation, and by a mechanical appliance. This consists of a poro-plastic felt jacket, and a head-piece connected by rubber door-springs, in such a way as to tilt the head in the opposite direction to that it occupied before the operation.

The lump may occur at any part of the muscle. The middle portion of the belly of the muscle is affected most commonly. This was so in thirteen of my cases; of the rest, the lower part was affected six times, the upper three times, and in one the swelling appeared to be limited to the sternal head.

The period at which the swelling was noticed was variable. It was observed as early as the second, third, seventh, eighth, and ninth days, and as late as the fourth or fifth week; but the most usual period was the fourteenth day, which is about the period when mothers in poor circumstances begin to attend personally to their babies. The most probable theory of the causation of the swellings is the original one of Dieffenbach, who in 1830 attributed them to stretching of the muscle and tearing of the blood-vessels during parturition. This theory has been strongly corroborated by the observations of Dr. Herbert Spencer ("The Journal of Pathology and Bacteriology," May, 1892), who met with haemorrhage into the sterno-mastoid muscle in fifteen bodies of infants who were either still-born or died shortly after birth; and in one case, which was examined microscopically, the muscular fibres were found ruptured in many places, and blood was seen to be effused in large amount between the fibres. The appearances presented by the sterno-mastoid muscle in some cases of torticollis lend support to the theory which connects the deformity with laceration of the muscle at birth. In one case in which I divided the belly of the sterno-mastoid muscle about midway between its origin and insertion there were a few apparently normal muscular fibres on the surface, but the central portion of it was composed of dense fibrous tissue, which had the consistence and the bluish-white tint of the tendo Achillis. A similar condition has been described by other observers, particularly by J. Vollert ("Centralblatt für Chirurgie," 1890, No. 38). Volkmann said that in many severe cases no changes were visible in the muscle—there was no scar or interruption like a tendinous intersection. Vollert corroborates this in one case. In two others there were muscular fibres like those of normal muscle, but they were less numerous, and they were smaller than natural. In some parts the muscular fibres lay close together, but in others they were separated by fine connective tissue. There was no fatty degeneration of the muscular

fibres. In one case the muscle was completely replaced by strong tendinous fibres.

Mikulicz, ("Centralblatt für Chirurgie," 1895, No. 1), who has in seventeen cases of wryneck excised almost the whole of the muscle, states that the whole of the sterno-mastoid is involved in a chronic inflammatory process, which he attributes to compression of the muscle during labour rather than to laceration of it.

There is a tendency to replace the old name of congenital tumour of the sterno-mastoid by that of haematoma of the sterno-mastoid. It is doubtful whether this change is a good one, for there is, so far as I am aware, no evidence to show that there is ever a circumscribed collection of blood. At the most there is only diffuse extravasation of blood amongst the muscular fibres. Moreover the swelling at the time when it is generally observed, viz. about the fourteenth day, has no resemblance to a haematoma. Its characters depend on the chronic inflammatory changes which supervene upon the laceration of the fibres and the extravasation of blood amongst them—in fact, on chronic myositis. My cases were seen first at various periods between three weeks and three months. In all there was a hard mass of almost cartilaginous consistence. It was strictly limited to the sterno-mastoid muscle. The surrounding tissues appeared quite normal. The mass itself was generally well defined in all directions, but sometimes irregular bands of less dense consistence could be traced into the muscular tissue above and below the main swelling. The swellings could be moved laterally, but not from above down. As a rule the head was held quite straight, and the function of the affected muscle was not obviously impaired. In eighteen cases the head was held quite straight, and with the exception of the swelling itself the two sides of the neck were quite symmetrical. In five cases the head was held a little to one side—towards the affected side, but in only one case did the freedom of movement appear to be at all impaired. As a rule the swelling did not appear to cause the child any discomfort. In three cases the lump seemed to be slightly tender to the touch.

In ten cases the date of total disappearance of the lump was noted. The usual date of complete disappearance was the sixth or seventh month. In one case no trace of the lump could be detected

when the child was three months old. On the other hand, in two cases the swelling did not disappear until the twelfth month.

It has only been possible to follow up fourteen cases; two had died, one when two years and ten months old, and the other when about ten months old, but no deformity had been noticed in either. The remaining twelve cases were examined. In only two cases was there any contraction of the affected muscle, and in those the contraction was extremely slight. One was three years old, and the other ten months old. In each the head was held a little to the affected side, the muscle was tighter and more prominent than its fellow, and the measurement from the ear to the clavicle was $\frac{1}{2}$ inch less on the affected side than on the other. In the remaining ten cases there was no deformity and no increased tension of the affected muscle. At the time of the observation three cases were between three and four years old, three cases between two and three years old, two cases between one and two years old, and two cases eight months old.

These observations show that in the majority of cases of so-called congenital sterno-mastoid tumours no permanent injury of the muscle results. On the other hand, Mr. Clutton, Mr. Edmund Owen, Mr. Raymond Johnson, and Mr. D'Arcy Power have observed cases in which congenital tumours of the sterno-mastoid muscle have been followed by torticollis, and the last-named surgeon has collected thirty cases (Mr. Clutton's, Mr. Willett's, and his own) which were followed up. Eleven had wry-neck; in two of the cases it was extremely slight, but in four it was so marked as to necessitate division of the tendon. Mr. Power has also collected from English and foreign literature 106 cases of congenital haematoma, and finds that wry-neck occurred in at least 21 of them. In two cases of well-marked torticollis upon which I operated last year there was a clear history of the affected muscle having been the seat of a hard swelling, which gradually disappeared in the first few months of life.

Cold Baths in Delirium Tremens.—Dr. Letulle recommends immersion of the body in water at the temperature of 64.4° F. The head should be cooled by large waves of water. The bath should last eight, twelve, or fifteen minutes according to the reaction of the patient.

(*Medical Record.*)

A CLINICAL LECTURE

ON

ENTERIC FEVER,

Being an Account of his own Illness.

Delivered at St. George's Hospital, June 1, 1896, by

JOHN CAVAFY, M.D., F.R.C.P.,

Physician to the Hospital.

GENTLEMEN,—Enteric fever is so constantly under our observation, and is so variable in every way—in its severity, in its symptoms and duration, in its complications, and in the sequelæ which may follow it—that every case, however simple, mild, and apparently unimportant, may furnish us with matter worth consideration. I venture, therefore, to give you a short account of a case, which although it affords little, if anything, that is novel, possesses at least this unusual feature, that I was myself the patient! I am thus enabled for the first (and I hope the last) time to give you a brief description of a case from the patient's point of view. In endeavouring to lay before you some of the more salient points connected with its onset, course, and subsidence, I am afraid that it will be impossible for me to avoid an appearance of egotism, and you may perhaps think that I am giving more prominence than they deserve to many of the symptoms I shall relate to you, however interesting they may have been to me at the time of their occurrence. I fear that this is inevitable; but, after all, the accuracy of the clinical picture is thereby increased, and it may even be that one or two of the points I shall touch upon may be new to you.

Onset.—I must begin by telling you that I am 58 years old, and that therefore I am long past the age at which enteric fever is frequent. All authorities are agreed that it is rare after 40, and you will find this borne out by the cases admitted to our wards. The source of infection in any isolated case is in most instances extremely difficult to recognize, and my own forms no exception to the rule. I had, towards the end of December last, more than one case of typhoid under my care in the wards, and it is just conceivable that I may have derived infection from one of them. How unlikely this is I need not tell you. Nurses, it is

true, who are in constant attendance on typhoid cases do occasionally contract the disease, owing no doubt to the accidental omission of the necessary precautions ; but I cannot call to mind any instance of one of our physicians doing so. In searching, then, for some other source of my attack, it occurred to me that I had been exposed to a channel of infection to which attention has only recently been drawn, namely oysters. It is easy to understand that an oyster bed may be exposed to contamination by sewage, and that the molluscs may in this way become carriers of the specific virus. Now in my case oysters were eaten on January 3rd, and I remember that one of those that fell to my share had a decidedly unpleasant taste ; as I refer the first decided symptoms of my attack to January 17th, exactly fourteen days from this time, it is quite possible that this was the source of infection in my case, although I cannot prove it. No one else present at the same dinner-party was affected, but I was afterwards informed that the beds from which the oysters were supplied on this occasion were by no means free from suspicion, and that more than one case had occurred in which the same origin was considered probable. Be this as it may, the symptoms during the period of incubation were, for the earlier part, so slight as not to attract my attention, although I have been told that I looked pale and worn. It was not until two or three days before the definite onset that I myself felt the combination of vague discomfort and weakness to which the term "malaise" is commonly applied. On January 17th there was marked frontal headache, with muscular and articular pains, which were accompanied by a great sense of weariness, and complete loss of appetite. This last was so sudden and so strongly marked in my case, that I should feel inclined to attribute some importance to it as a symptom of onset. On the 18th the above symptoms, especially the loathing for food, were still more marked, but I went to the hospital as usual, finding great difficulty in getting through my work. On the following day, towards evening, as my condition remained the same, I used the thermometer for the first time, found that my temperature was 102.5° , and went to bed, this being as I judged the third day of the disease. I regret that I did not take my temperature before this, as I certainly should have done ; I am sure that early thermometry in symptoms

like those I have detailed to you, is most important and valuable, not only in diagnosis, but also in enabling the necessary treatment to be carried out at the earliest possible time.

Course.—The fever for the first ten days showed little variation, and was almost continuous, the highest point reached being 102.8° , and the lowest 101.3° . The evening rise and morning fall, so constantly noticed, were not observable, the morning and evening temperatures being often equal, or differing by fractional parts of a degree, and the morning temperature was not uncommonly the highest. On the eleventh day there was a fall of two degrees (from 102° to 100°) and from that time until the twenty-first day, on which a normal temperature was reached, the fever ran an irregular course. It varied from 99° , the lowest, to 103° on the evening of the nineteenth day, falling on the twentieth day to 99.4° , and to normal on the day following. The differences between morning and evening were often considerable, as much as two or three degrees, and the morning temperature nearly always the lowest. During this time, as will be seen later, I was troubled with the disagreeable complication of retention of urine, and perhaps some of the wide oscillations may be accounted for by the great discomfort accompanying this condition, and the measures adopted for its relief. The frequency of my pulse was hardly increased. It varied from 76 to 84, even when the fever was highest (103°), and it was only now and then that a rate of 90 to 104 was reached, usually after excitement or fatigue. It was firm and of good quality throughout, and dicrotism was not observed. I may perhaps mention here that I have long had an extremely well-marked *arcus senilis*, which was at one time thought to be indicative of fatty heart ; this, however, is a view which is now practically abandoned, as the condition may certainly exist without fatty change in the heart itself. My tongue was moderately furred, and I had no diarrhoea throughout, the motions being always formed, or nearly so. During the first twelve days there was some constipation, as many as five days having elapsed without action of the bowels, but usually they were moved more than once a day, three to five motions often taking place in the twenty-four hours. They became much more frequent later on, owing to a complication which I shall presently refer to. No blood was ever

passed. Characteristic *spots* were first noticed on the eighth day, and were found from that time appearing day by day, but were always few in number, and sometimes doubtful. *Abdominal distension* was moderate, and the *spleen* could not be felt. My *general condition* during the first fortnight or so was one of hebetude and apathy; I felt a stupid indifference to everything, and had only a dim comprehension of what was going on. The headache disappeared in three or four days, but was immediately followed by a curious *hyperæsthetic deafness*. I know that this contradictory term seems absurd, but it really expresses what I felt. I could not hear words at all plainly, even if the voice was considerably raised; all articulate sounds seemed to reach me muffled and obscure, perhaps partly owing to my mental condition; but the ordinary sounds of the sick-room were greatly exaggerated, and often caused me considerable annoyance. The noise of poking the fire, putting fresh coals on, &c., all seemed to me distressingly loud, and especially once I remember, the accidental fall of a poker in the fender, sounded like the din of artillery. The deafness rapidly disappeared, and had quite left me in about a week, but the extreme sensitiveness to sounds subsided more slowly, and was still perceptible during convalescence.

Complications.—These were mostly unimportant, although some of them seem to me unusual. One which I have not found mentioned by authors was a very transitory *acute conjunctivitis*. This occurred on two or three occasions during the continuance of the fever, invariably with a sudden onset, characterised by severe aching and smarting pain in the eyes, with profuse lachrymation. It always subsided after a few hours, but was certainly distressing enough while it lasted. *Tracheitis*.—Cough was a prominent symptom from the beginning, with frothy sputum at first, which rapidly became muco-purulent. No bronchitis could be detected on repeated physical examination, and no doubt the trachea was principally affected; some *pharyngitis* was also present. *Retention of urine*.—This unpleasant complication did not make its appearance until the eleventh day, and necessitated the passage of a catheter morning and evening. It was followed by slight inflammation of the neck of the bladder, accompanied by severe strangury at first, and later

by some diminution in control, with abnormally frequent micturition. Two curious results also came on about the same time. The first was *acute eczema of the scrotum*, which may have been due to reflex irritation. It yielded readily to simple local treatment. The second was a condition of *irritable rectum*, with much tenesmus and extremely frequent efforts at defæcation, reaching on one occasion the enormous number of sixteen motions in the twenty-four hours. These, however, hardly deserve the name, as the very smallest quantity of faecal matter in the rectum led to an immediate irresistible expulsive effort. This condition lasted three days only, and subsided without treatment. *Muscular rheumatism*.—Severe pain in the left loin and flank, greatly increased by movement, appeared on the eighteenth day, and continued very troublesome for about a fortnight, when it was suddenly and completely removed by the following accidental manipulation. In the course of bed-making (my temperature being then normal) I was rolled over by the excellent nurse who took care of me, first on one side, then on the other, and raised a little to allow fresh sheets to be passed. On one of these occasions I felt as if "something gave way" in the painful spot in the flank, followed by immediate relief, and it was possible for me for some days afterwards to obtain an unmistakable grating and rubbing sensation in the part by taking the deepest possible inspiration. It was exactly as if an adhesion had been broken down, due to lymph between two muscular surfaces, and I have little doubt that this actually occurred.

Relapse.—A normal temperature was reached precisely on the twenty-first day, and continued so for ten days, when a gradual rise began, until on the thirty-fourth and thirty-fifth days the evening temperature reached 102° . It then subsided after considerable oscillations, and fell to subnormal on the thirty-eighth day. The whole return of fever therefore lasted a week only, and perhaps hardly deserves the name of relapse. It was, however, accompanied by a return of conjunctivitis, by the appearance of one or two fresh spots, and by one most unpleasant and even alarming (to me) symptom, viz. a sudden paroxysm of intense *dyspnoea* lasting about ten minutes, which came on quite unexpectedly on my moving in bed, and subsided as suddenly. I

confess I thought of pulmonary embolism, and became much depressed, but my anxiety proved to be groundless. It was thought by Dr. Whipham and Dr. Pye-Smith, who most kindly attended me, that the intense difficulty of breathing (I have felt nothing like it before or since) was probably to be explained by sudden flatulent distension of the stomach or colon, pressing upon the diaphragm and hampering the heart's action. I must, however, note that there was no corresponding abdominal distension. I should say that the slight relapse I have described could not be accounted for by any imprudence on my part. The diet continued entirely fluid, and no other change of any kind was made.

Convalescence.—Although a subnormal temperature was reached on the 38th day, there was some slight oscillation for another week, the evening temperature rising to 99° or a little over. It was not until the 45th day that a permanently subnormal or sometimes normal temperature was finally reached ; it was on that day that I was first allowed a little breadcrumb, and from that point convalescence became gradually established. I do not know that there is anything unusual in the very slow and gradual return of strength ; convalescence is always a tedious matter in typhoid, and it is obvious that time is required for the atrophied muscles to regain their bulk. To this day I find that I am very easily tired, and that I often feel a curious difficulty in beginning to walk, the legs feeling stiff, weak, and awkward at starting ; this, however, wears off in a few minutes. I should say that the ravenous appetite so often seen in younger typhoid convalescents as soon as the fever is over, did not show itself in me until I was up and moving about the house, and even then moderately. The skin desquamated very profusely ; more so, I think, than in most cases I have seen, the thick epidermis of the soles especially coming off in large flakes. The new epidermis formed was certainly thinner than before, the skin feeling unusually soft and satiny. This perhaps accounts for my feeling "footsore" even after the shortest walk, and may partly explain the tenderness of the toes described by Handford. It is probably owing to the free desquamation that occurred, that a patch of *erythrasma*, which had long been present on my left thigh, spontaneously disappeared, the fungus being doubtless carried away in the shed epidermic scales. The tongue was raw and sore for some time owing to loss of epithelium, so that the contact of hot liquids, pungent condiments, and wine, was rather trying owing to the smarting they caused. The transverse marking and ridging

of the nails so often seen after exhausting illnesses are very distinct.

Treatment.—So far as drugs were concerned, none were employed for the fever itself ; a little morphine and ipecacuanha for the cough, tincture of henbane, benzoate of sodium, and later fluid extract of sandal-wood for the cystitis, were all that was required, and the treatment consequently resolved itself into complete rest in bed, and fluid food. This consisted chiefly of milk, of which two pints were given in the twenty-four hours, and one pint of beef-tea, or various other broths, half a pint of nourishment of one or other kind being administered every four hours throughout the day and night. It was curious that during the fever I developed a great liking for milk, which I greatly preferred to broth of any kind ; in my usual health I am quite indifferent to it, and never take it in any quantity. I am convinced that the daily amount of food taken, three pints in all, was amply sufficient, as when I took even a little more, which happened once or twice, the result was to cause a good deal of uneasiness from flatulence. No doubt the amount of food and frequency of administration should vary according to the age of the patient and the severity of the case, but I am convinced that the tendency is on the whole to give too much. Any over-feeding is in my opinion to be strongly deprecated, and may obviously do harm by increasing peristalsis, diarrhoea, and abdominal distension. *Stimulants* were tried once or twice in small quantities, more as a matter of custom than because they were required ; it was found, however, that they increased my bladder discomfort considerably, and were therefore at once discontinued. I practically took none at all during the fever, and none during early convalescence, returning to a moderate amount only by slow degrees. I may say that I had a strong dislike to them throughout, and that their absence was no privation.

Conclusion.—As you have seen, I had a mild attack of typhoid fever without serious complication, and the question arises whether my age had anything to do with this. I was informed by a young medical friend that all senile cases (among which he was good enough to include me) did well, as they had no Peyer's patches to speak of, owing to the atrophy of age. If this be so I am not so old as I look, as I had at least enough to provide for a relapse. But my own experience is that cases occurring after 40, which are rare, as I have already said, are by no means always favourable. I remember a fatal case in a lady over 70 years old, and recently had a case under my own care, aged 49, in which dilatation of the heart led to a fatal result a few months after the fever was over. In cases which recover, like my own, I think restoration to health is slower and more tedious than in younger subjects.

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A CLINICAL LECTURE

ON TWO CASES OF

UNILATERAL PARALYSIS OF THE OCULAR SYMPATHETIC.

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ALTHOUGH precise symmetry of the pupils is, as Hutchinson has observed ("Different states of the pupil," "Brain," vol. i. page 8) the exception rather than the rule, yet any marked and persistent inequality is a symptom which always requires careful investigation; for apart from local, and perhaps serious conditions, such as glaucoma or paralysis of the third nerve and ophthalmoplegia interna, which may cause it, it may betoken grave and unsuspected disease of the intra thoracic organs, or of the brain or spinal cord, and its surroundings. One fallacy of observation in the matter may be mentioned here. It is that in people whose pupils are naturally unduly mobile, dilatation of the one on the side furthest removed from the light, may be often observed, and may be considered a morbid condition, but if the eyes are equally exposed to light or shade, the inequality at once disappears. I shall not discuss in detail the differential diagnosis of the various states which give rise to inequality of the pupils, but shall confine myself to the consideration of two cases in which it and other symptoms are apparently due to paralysis of the ocular sympathetic. It will be seen, however, that in one case at least, and perhaps in both, the condition is rather attributable to disordered function of the ocular sympathetic than to actual paralysis. One eye only is afflicted in each case; but I may mention that *double* ocular sympathetic paralysis may occur, and may easily escape notice, as in

this instance there will be no noticeable difference between the two eyes, and the paralysis will only attract attention when one eye has recovered. Cases of sympathetic paralysis are perhaps more familiar to ophthalmic surgeons, or to surgeons who have to treat cases of direct injury to the cervical sympathetic, than to the physician or the general practitioner; and perhaps it is to these that the following cases may be interesting. The patients are a medical man, aged 38, and a girl employed in an optician's shop, aged 17. Both exhibit the signs of paralysis of the ocular sympathetic, first accurately described by Claude Bernard ("Comptes Rendus," 1862, t. lv. p. 382), namely:—(1) slight ptosis; (2) narrowing of the palpebral fissure; (3) slight recession of the eyeball; (4) contraction of the pupil. The right eye is affected in both cases. The symptoms in the man have been noticed for fifteen years. A photograph taken at this time first drew his attention to the condition. In another photograph taken three years previously it is not apparent. In the girl's case, the symptoms have been present for about six years. Some months previously she had received a severe blow, the scar of which remains on the root of the nose. Her nasal cavities are healthy now, and there is no history of nasal affection following the injury. The man, when eight years old, suffered from slight concussion of the brain after falling on the ice and striking his left occiput. From this time until the age of twenty he was subject to frequent left-sided migraine, always preceded by complete left hemianopia. No such headaches have occurred since. The girl complains of frequent right supra-orbital headaches, but they do not appear to resemble those of migraine. Otherwise, the general health of both patients has always been good. Taking these symptoms in detail, the ptosis is slight in character, the upper eyelid never obscures the pupil as in the case of paralysis of the third nerve. Neither, as in the latter complaint, is the frontalis muscle wrinkled on the affected side from the effort to elevate the lid. It is also less complete than in people with hysterical ptosis. There is no quiver-

ing of the eyelid or resistance to passive attempts at elevation. The narrowing of the palpebral fissure is due not only to falling of the upper lid but also to rising of the lower, which appears to be at a higher and more horizontal level than that on the opposite side. The recession of the eyeball is slight but obvious. There is no difference in the intra-ocular tension of the two eyes in either case, and there is no reddening of the conjunctiva or lachrymation. The right eye looks smaller than the left, and this gives the impression that the whole right side of the face is smaller than the left. But there is no actual hemiatrophy. There is no rise or fall of temperature, no flushing, pallor, or sweating, no abnormal dryness on the right side of the face or neck, and no history of such symptoms. There is no weakness of any of the *motores oculi* muscles. The pupillary contraction is moderate in both cases; it never amounts to extreme myosis, such as is seen in cases of tabes. Hutchinson insists that, in cases of sympathetic paralysis, simple inability to dilate, and not contraction of the pupil, is the usual condition ("Brain," Vol. i., p. 11). If both our patient's eyes are equally exposed to light, the right pupil remains smaller than the left. If the left is exposed, and the right shaded, the latter dilates slightly, and becomes equal to its fellow for a moment. But then, consensual dilatation of the left pupil occurs, and the inequality is again noticeable. If the left pupil is shaded, consensual dilatation of the right pupil occurs, but is slight; and lastly, if both pupils are shaded, the left is far more dilated than the right.

Irritation of the side of the neck by means of a powerful faradic current does not cause dilatation of either pupil. This superficial reflex is absent in both cases. It is doubtful, however, if any significance may be attached to the fact any more than to the common absence of other superficial reflexes in normal people. Re-action of the right pupil to accommodation is in both cases good, and perhaps slightly more active on the right than on the left side, both patients are slightly myopic, vision is equal in the two eyes.

Variations of Symptoms.—In both cases the symptoms show marked variation. They are always more apparent when the patients are fatigued, or out of health in any way. At times they are almost unnoticeable. In the girl's case,

they are most pronounced on going to bed and on getting up. In the case of the man, a curious alternation is noticed. At times the symptoms are replaced by those of the opposite condition, namely, irritation of the ocular sympathetic. The right eye appears even more widely open than the left, the upper lid being elevated as in the condition common in Graves' disease, and described by Stelwag. There appears to be slight proptosis, and the right pupil is slightly more dilated than the left. This has been observed mostly on rising in the morning, the change is extremely transitory, soon giving way to the usual or opposite condition described. The inference drawn from these alternations must be that disordered function rather than paralysis of the sympathetic is present. The occurrence of such alternations is noteworthy, for in some cases it may cause difficulty in deciding whether sympathetic paralysis of the one eye, or sympathetic irritation of the other, is the cause of the symptoms. The affected pupil in paralysis dilates sluggishly in shading; in irritation it contracts equally slowly on exposure to light.

Action of Mydriatics.—In both cases the right pupil dilates fully and normally under atropin, but there is a marked difference in the action of cocaine in the two patients. Two drops of a 5 per cent. solution of cocaine in the man's right eye produce in five minutes distinct elevation of the upper eyelid, and widening of the palpebral fissure. This effect passes off in a few minutes; the pupil then begins to dilate, the dilatation attaining its maximum in about one and a half hours, and not disappearing until eighteen hours have elapsed. Accommodation at the time of the dilatation seems completely paralysed. A similar application to the girl's eye produces no effect whatever, either in dilating the pupil, or in widening the palpebral fissure. This observation was made by Dr. Rayner Batten, who sent the patient to me, and was confirmed by the patient, and also by myself. This difference in the reaction to cocaine is most important. The drug acts by stimulating the peripheral terminations of the sympathetic nerve fibres in the iris, and the absence of reaction shows that in the girl there must be some degeneration of these terminal fibres, whilst its presence in the man's case shows that they are not so degenerated. As remote and possibly exciting causes, the injury in both cases

has been mentioned. As to the present possible causes, the girl has some slightly enlarged glands in the right side of the neck, and the right external carotid in the man always appears to be slightly larger, and to pulsate more forcibly than the left. It is possible that either of these conditions may produce the symptoms by pressure on the cervical sympathetic; but there are reasons against this view. Both patients have suffered from neuralgia of the fifth nerve, due to carious teeth. It being possible that the sympathetic fibres, which run with the fifth nerve, might have been affected from this cause, several of the girl's upper teeth on the right side were removed, and others stopped by Mr. Russell Barrett. After these operations, the condition at first greatly improved, but ultimately, although everything had been removed which could give rise to the trouble, all signs of improvement disappeared. Mr. Barrett has met with a case in which very marked ptosis was present; some carious teeth were extracted under an anaesthetic, and when the effects of the anaesthetic had passed away the ptosis disappeared entirely. Lauder Brunton noticed constant spasmotic twitching of the right upper eyelid after removal of a bicuspid stump from that side of the jaw. It lasted until the wound in the gum had healed ("Disorders of Digestion," 1866, p. 91). Such cases show that dental affections may cause ocular troubles; but the man only retains one of his right upper back teeth, and this alone seems hardly likely to account for symptoms lasting for so many years.

REMARKS. *Anatomical.* — Anatomically the sympathetic supplies the unstriped muscles in the neighbourhood of the fissura orbitalis, and also probably the similar unstriped muscles which exist in the upper, and especially in the lower eyelids. Müller ("Verhandlungen der Würzburger phys. med. Gesellschaft," 1859, Bd. ix., p. 76) traced the nerve supply of these muscles to the sphenopalatine ganglion, but Prévost demonstrated that irritation of the cervical sympathetic still produced protraction of the eyeball after extirpation of this ganglion.* Probably they are supplied by the sympathetic plexuses which accompany the blood vessels to this area. Sappey (*Sitzung der Pariser*

Académie des Sciences

, 21st Oct. and 18th November, 1867) has described other unstriped muscles in the orbital aponeurosis. It is inferred that these and also the palpebral muscles are supplied by the sympathetic, because proptosis with widening of the palpebral fissure are produced by irritation, whilst narrowing of the fissure and retraction of the globe follow section of the sympathetic. The sympathetic is held by Remak ("Deutsche Klinik," 1855, No. 27 p. 294) to supply tonic force to the voluntary ocular muscles, called levator palpebræ superioris, retractor plicæ semilunaris, and orbicularis oculi. According to Romberg the internal strabismus which follows division of the sympathetic is due to want of tone in the external rectus which receives fibres from the ascending branches of the uppermost cervical ganglion, as well as from the sixth nerve, whilst Schiff holds a similar view as regards the oblique muscles, for after division of these, irritation of the sympathetic no longer produces exophthalmos (Eulenbergh and Guttman, "The Sympathetic Nerve System," 1879, p. 5.). The sympathetic probably ends on the surface of the cortex cerebri, for stimulation of certain cortical centres situated in the lower part of the second frontal convolution, the angular gyrus, superior temporal gyrus, and perhaps occipital lobe (Ferrier, *Cerebral Localization*, "Lancet," June 14th, 1890), causes the eyes to become widely opened, the pupil dilated, together with deviation of the eyes towards the opposite side. It is conceivable that destruction or lowered vitality of these same centres may produce the opposite train of symptoms, such as are present in the cases now under consideration. As regards the action of the sympathetic in producing dilatation of the pupil there is some difference of opinion. That dilatation is due to the influence of the *vaso motor* sympathetic fibres is disproved by the fact that irritation of the sympathetic causes marked dilatation in the pupil an hour or more after death (Gaskell, "Journal of Phys.," vii, 1, p. 38). The presence of true dilating muscular fibres in the iris has been disproved by Schwalbe, Fuchs, Jessop, and others. Gaskell regards dilatation of the pupil on the stimulation of the sympathetic as brought about by "inhibition of the tonic contraction of the sphincter iridis in conjunction with the elasticity of the posterior limiting membrane or outer part

* *Recherches sur le ganglion spheno-palatin*, "Arch. de Phys.," 1868, p. 221.

of the iris, in consequence of which the pupillary edge of the iris naturally falls back when the sphincter is relaxed" (*op. cit.*). The mechanism by which the pupil is dilated is of course reflex. The centres concerned are:—

1. Those situated below the floor of the corpora quadrigemina, external to the nucleus of the third nerve (Henson & Volckers).

2. The centrum cilio spinale superius and also cilio spinale inferius in the cord. The lower level of these centres is not below the origin of the second dorsal spinal nerve; their upper levels are unknown, but probably they extend to—

3. *Cortical centres* (already mentioned).—Bevan Lewis* supposes that a strand of fibres connects the motor oculi nucleus for constrictor pupillæ with the dilator centre beneath the corpora quadrigemina. The dilator centre itself being in connection with the vaso-motor tract in the medulla. "Such connecting fibres," he says, "would have to be regarded as inhibitory; their excitation through optic stimuli inhibiting the normal action of the cilio spinal tract. Shading the eyes in normal subjects would reduce such activity and favour dilatation. Division of such fibres would of course act likewise, whilst disease lower down in the cilio spinal region would induce a myosis through unrestrained activity of the motor oculi." The centripetal fibres are supplied by all the sensory nerves from the fifth cranial to the last dorsal. The cilio-spinal tract passes down the cord to the centrum cilio spinale inferius of Budge (*Über der Bewegungen der Iris*, 1855). Thence motor fibres emerge with the anterior root of the second dorsal nerve ("Gaskell's Journ. of Phys.", vii., 1, p. 38) joining the cervical sympathetic, with which they pass on to the cavernous plexus, the Gasserian ganglion, ophthalmic division of the fifth and its nasal branch.

Filaments from the cavernous plexus form the sympathetic root of the ciliary ganglion, whilst the long ciliary branches of the nasal nerve join the short, which proceed from the ganglion itself, to supply the iris and the ciliary muscles. The lenticular or ciliary ganglion itself is not necessary for dilatation of the pupil, for Andamuk demonstrated that after extirpation of this ganglion

stimulation of the cervical sympathetic still produces dilatation of the pupil. As already stated, Prévost has proved that the sphenopalatine ganglion is not essential to the working of the orbital and palpebral unstriped muscles. The site of lesion in these cases may therefore be:

1. In the sensory nerves generally. This may be disregarded as not probable or possible in the absence of other symptoms of such widespread disturbance.

2. In the cilio-spinal ganglia of Budge. Affections of these centres in the cord are commonly supposed to cause the well-known oculo-pupillary symptoms of tabes dorsalis. But in the present cases such affection may be excluded, in the entire absence of any symptoms pointing to the disease.

3. The involvement of the nerves (upper two dorsals) by tumour or aneurism may also be excluded.

4. Implication of the cervical sympathetic in the neck by the enlarged cervical glands in the case of the girl and by the enlarged carotid in the case of the man are probably not the causes, for if they were, the symptoms would depend on pressure paralysis. The glandular enlargement appears insufficient to cause this, and a difference in the size of the two carotids often exists without producing symptoms of pressure paralysis. Moreover, were this so, they would hardly have continued for so many years without evidence of implication of the vaso-motor fibres of these nerves, instanced by changes of temperature, flushing or sweating, pallor or dryness of the side affected. All such symptoms are absent now, and there is no history of their occurrence at any time.*

5. The lesions, therefore, are probably intracerebral. We have before excluded from the sphere of action reflex dental disturbance of the sympathetic fibres which run with the fifth nerve. Were the Gasserian ganglion the site of mischief the symptoms would not be confined to the eye. The lenticular ganglion and also the sphenopalatine ganglion have also been excluded, on the ground that their destruction does not impair the power of dilatation and also because the

* ("Brit. Med. Journ.", vol. i., p. 1082, 1896) "Ocular Symptoms of General Paralysis."

* It is fair, however, to state that in cases of division of the cervical sympathetic the vaso-motor symptoms produced at first are apt to disappear entirely, whilst the oculo-pupillary effects remain.

orbital muscles are affected, which they do not supply, but which are probably supplied by the vascular sympathetic plexuses. The sites which remain are the cavernous and carotid plexuses, the centres beneath the corpora quadrigemina and their connecting fibres with the other ocular centres in this position, and finally the centres of the cortex cerebri. The dilator centres beneath the corpora quadrigemina apparently only subserve the function of dilatation of the pupil, but in both cases it is not only the pupil that is affected, but also the eyelids and the orbital muscles are involved. Were the affection entirely confined to the pupil one might imagine that the dilator centre alone was the seat of the mischief, yet a nuclear lesion wide enough to produce the whole symptoms present would probably involve the other centres close by, viz. the oculo motor and the centre for accommodation. But there is no evidence that these are affected. Neither would a lesion involving the hypothetical connecting fibres between the oculo motor and dilator nuclei account for all the symptoms. The lesion, therefore, is probably not of these fibres nor of the centres themselves beneath the corpora quadrigemina. It will be remembered that in both cases there is a well-defined history of injury which preceded the symptoms. In the girl's case the injury was to the root of the nose, in the man's to the left side of the occiput. It will also be remembered that a great point of distinction between the two cases is that in the girl there is complete absence of reaction to cocaine, whilst reaction is normal in the man. This absence of reaction denotes, as we have seen, degeneration of the terminal fibres of the sympathetic in the iris and orbital muscles. An injury to the sympathetic in the neighbourhood of the cavernous sinus and carotid plexus may account for the degeneration present and for the presence of the symptoms. Such an injury may have well been produced by the blow on the root of the nose recorded.

I would, therefore, suggest that the symptoms in the girl's case depend on an injury to the sympathetic, probably in the neighbourhood of the cavernous sinus. The exact site of the lesion cannot be defined. One can only assume that it is deep enough to involve both the sympathetic fibres which supply the orbital muscles, and also

the ciliary nerves, which dilate the pupil. The amount of reaction on shading, which still exists, and the curious remissions and alternations of the symptoms, show that the nerves cannot be wholly degenerated in either case. Or, perhaps, in the girl's case the sympathetic root from the cavernous plexus to the lenticular ganglion is injured. This would affect the short but not the long or nasal ciliary nerves, and might account for the preservation of accommodation.

In the man's case, there is no evidence of such degeneration of the terminal sympathetic nerves, so a different explanation must be sought. There is a history of frequent left-sided headache, with hemianopia following the injury to the left occipital region, and preceding the sympathetic paralysis. Such symptoms point to cortical disturbance. Cortical irritation may produce symptoms resembling those of irritation of the cervical sympathetic, and destruction of certain cortical areas will be followed by symptoms of paralysis of the cervical sympathetic. It is true that in the man's case there is no history of symptoms of ocular sympathetic irritation prior to the onset of paralysis, yet the curious alternations noticed since between symptoms of irritation and those of paralysis of the ocular sympathetic, together with the absence of evidence of terminal degeneration of the nerve fibres, suggest a cortical origin of the mischief. The history of migraine and left hemianopia is in favour of this view. It is probable, therefore, that the cortical injury produced by the fall on the ice, many years ago, may at first have produced symptoms of cortical irritability, instanced by the migraine and hemianopia before mentioned, and that in time, irritability of the centres involved may have given way to lowering of functional activity, but not to absolute destruction of the area involved. As to treatment in the man's case, the condition affords him no inconvenience, except that he is unable to use his right eye for the ophthalmoscope or microscope. In the case of the girl, she is alive to the impairment of her personal appearance, and desires that something should be done. A course of galvanism and judicious application of cocaine is now being carried out.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM

BY

JONATHAN HUTCHINSON, LL.D., F.R.S.

Reported by J. T. CONNER, M.D.

An Undiagnosed Eruption (Lupus?).

A woman, aged 50, sent by Dr. Woodd Walker, showed on the inner surface of the knees some rounded patches, about the size of sixpences. They were of a dusky red colour, in fact exactly that of a syphilide, very slightly elevated, and a little scaly. There were four on the left knee, and one on the right. She had noticed them for fifteen years, and others had been present on the thighs which had now disappeared. These were not attended with any disturbance of health now or at any time.

Mr. Hutchinson thought the case was probably a variety of lupus. But the only cases he had seen at all resembling it were two shown some time ago at the Demonstrations—one an old woman, Mrs. M—, and the other a middle-aged man, a station-master.

Lupus Vulgaris.—Two Cases.

The first was a woman, aged 45, brought by Mr. G. W. Sequeira. The disease consisted of a band extending across the front of the neck from ear to ear, and invading the conchæ. The central part showed the vascular "veal skin" scar, whilst the edges were formed of typical "apple jelly" growth divided into promontories. There were many large scales on the surface, which the patient aptly compared to those of a fish. This condition had been termed *lupus exfoliativus*, and wrongly considered a variety; but it was only a stage. The disease commenced when she was 7 years old, and had been slowly spreading ever since. In conformity with the law laid down by Mr. Hutchinson, it remained localised, and showed no tendency to affect distant parts. There was no history of any form of tubercular disease in the patient's family.

The second was a woman, aged 51, brought by Dr. Fletcher. The disease was located in almost exactly the same place as in the previous case, but

it contrasted with it in every other respect. It had begun in middle life, a year ago, and not primarily as a skin disease, but secondary to suppurating strumous disease of the submaxillary glands. These had been attacked one after the other during the last thirteen years. The lupus began as a small red patch, in the middle line, in one of the scars, and gradually extended from this on either side. The scars left were whiter, healthier, and more puckered than in the previous case. No apple-jelly deposit could be seen except a little below the left ear. Thus the case was rather one of scrofuloderma complicated with lupus. It was most unusual to find lymphatic tuberculosis extending to other tissues, or lupus vulgaris complicated with any other tubercular manifestation. There was a family history of tuberculosis—three of the four brothers of the patient had died of consumption, and one child had an "abscess in the groin."

Dermatitis Herpetiformis (?).

The patient was a man, aged 21, sent by Dr. McRae. Three months ago, whilst in good health, an erythematous eruption appeared. It began simultaneously at the ends of the four fingers on each hand, beneath the free margins of the nails. It quickly spread up the arms, and then became general. In about a fortnight the chest and soles were involved, and a week later the back. Two or three days later, blisters occurred first on the back, then on the chest and abdomen, then on the arms, and last on the legs. They lasted altogether about a month, coming out in "crops," distributed in groups "like bunches of grapes." The largest were "about the size of half-crowns" on the arms, being formed by coalescence of smaller ones. He had been treated with arsenic since the first week. When he presented himself for demonstration, the vesicle and bullæ had entirely disappeared, leaving a general, dusky erythematous eruption desquamating on the extremities, and diffuse only on the hands. On the chest it was finely mottled, like the scarlatina rash. On the extremities large areas were unaffected—for example, the middle two-thirds of the thighs, and spaces the size of the palm below the elbows. It was most severe at the large joints and on the loins. He had been losing weight, and felt ill somewhat, which was perhaps due to the arsenic.

Mr. Hutchinson said that, though nothing characteristic was to be seen, he saw no reason to object to the diagnosis which had been made—Dermatitis Herpetiformis—a name which was now, he thought, too widely applied. A number of different constitutional skin affections were included. He had had portraits executed, and had described the disease as Pemphigus Herpetiformis before Duhring did so as Dermatitis Herpetiformis. He still preferred the former name, as it expressed a real relationship to the Pemphigus group. The disease was characterised by the appearance of groups of vesicles, each group exactly resembling one of herpes. Arsenic usually would cure an attack, but relapses would follow. Like the other diseases for which arsenic was a specific, nothing was known as to the cause, and it attacked persons in perfect health. It was seldom fatal, but he had recently seen one so in two months. Sometimes it began exactly like psoriasis. It did so in the case mentioned, and had been so diagnosed by an able specialist. The case now shown was not so well characterised as the more chronic forms. He had never seen the whole hand involved in the inflammation before. Perhaps the cause was that it was a chilly hand of feeble circulation. The patient presented himself again three weeks later. He had stopped the arsenic, and had put on weight, and began to feel quite well. The eruption disappeared entirely, except on the palms, ten days ago. But six days ago it reappeared, first on the back near the shoulders.

The condition of the hands was peculiar. On the palms, which were universally red, were many little round spots, which looked like drops of tallow, the size of split peas, but confluent in many places. He said that these spots sometimes "came up," they swelled, but never developed blisters. The second eruption was freely out in the form of large broken rings, which were slightly raised at their edges, and here and there, but not usually, attended with small vesications. The erythema was easily discharged by pressure. The skin was left with a brownish stain where the former eruption had ceased. On the forearms, and on part of the trunk, there was a faint mottling of white spots, such as on the palms. The rings on the back were very large, making patches as big as the outspread hand. He said that the second eruption irritated him so much that he could not lie down in bed.

Mr. Hutchinson said that he could not give a nominal diagnosis here. He would describe it as an urticarious erythema, with threatened vesications in the palms and forearms. He compared it with the cases he had described as "Tebb's Eruption."

Multiple Tubercular Subcutaneous Abscesses.

The patient was a female infant, aged 19 months, brought by Dr. Diver. She showed a number of small hard nodules, about the size of peas, distributed irregularly beneath normal skin, and a large one on the ulnar side of the back of the right hand, adherent to the overlying skin, which was congested. It was about the size of a cherry, and distinctly fluctuated. The smaller ones were in the following situations: the inner side of the right wrist, the inner side of the right knee, the back of the right hand, the inner side of the left wrist. The child had always been in good health, and there was no obvious cause of the disease; but the father had died of consumption when she was six months old.

Mr. Hutchinson considered it to be a case of multiple tubercular subcutaneous abscesses—a form of tuberculosis which attacked infants, often apparently in good health, and which ended in perfect recovery, and showed no tendency to involve other structures, except possibly the joints. He compared it with a case sent by Dr. Abraham, already published (*CLINICAL JOURNAL*, 1895, May 8th, p. 30). A fat, healthy infant, aged 7 months, showed about twenty subcutaneous lumps, varying in size from a pea to a cherry. The family history of consumption was of interest. There was, no doubt, inheritance of tendency, at least to the tuberculosis. But (as he had often pointed out in the case of other diseases) there was "transmutation in transmission," in regard to the tissue attacked.

Pigmentation of the Lips and Mouth.

The patient was a woman, aged 43, who was under the care of Dr. Knowsley Sibley for tachycardia. Four years ago she noticed a black spot on the centre of the prolabium of the lower lip. This was followed by others on the lip and in the mouth, which had increased in size of late. Both prolabia were covered with ink-black spots, varying in size from a pin's head to a pea.

They were, too, on the mucous membrane of the cheeks and hard palate, but not on the gums. Also, a year ago, a spot appeared on the left side of the forehead, which was now the size of a pin's head. The patient was of a dark complexion, much freckled about the face, and in early life very liable to bronze on exposure to the sun.

Mr. Hutchinson said that this was a rare form of pigmentation, liable to affect persons of dark complexion. He had seen a clergyman, aged 62, with precisely the same patches. He also compared the case with the twins brought by Dr. Conner, shown several times at the demonstrations, of which a portrait was produced. In them much the same area was attacked, but otherwise their cases differed in several important respects. It was much rarer, because the pigmentation began in childhood, and further of great interest, because it attacked twins at exactly the same time. The formation of pigment patches, which occur in senile periods—"senile freckles"—was also mentioned. These were often followed by the formation of cancer in the adjacent skin. The pigment spots themselves did not become cancerous, but the process was no doubt infective, in the sense that each freckle was liable to produce others, as well as to extend, forming a melanotic stain. This might extend on to mucous membrane. In one remarkable case he had published ("Archives," iii, 32) it had done so from the lower eyelid to the conjunctiva, and even the cornea, producing a sepia tint of the arcus senilis.

Pityriasis Rosea simulating Syphilis.

The patient was a young woman, aged 21, sent by Dr. Sibley. She was covered with a general, copper-tinted, slightly scaly eruption. It consisted for the most part of macules, varying from a pin's head to the cut surface of a split pea, but a few small papules were to be seen here and there. The face and palms were exempt. On the inner surface of the right upper arm was a slightly elevated scaly patch, about the size of a sixpence. This was noticed first ten days ago. Four days later the general eruption appeared, attended with considerable itching. The patient had been married six weeks, and stated that she was not suffering from any affection of the genitals. The throat was normal.

Mr. Hutchinson said that the eruption, which

was to some extent polymorphous, differed in no respect from a syphilitic exanthem. In fact, he would not yet positively exclude that diagnosis. But the following facts tended to establish that the case was pityriasis rosea:—

There was no history of a primary sore, but of a "parent patch," existing alone for four days. The throat was normal. The face was unaffected.

NOTES FROM THE CLINIC

OF

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Assistant Physician to University College Hospital.

Lymphadenoma.

THIS woman has been ill for about four months. She has a lump in her neck on the left side, her age is 42, and you notice also that there are lumps in her left groin. These lumps are numerous, and the patient does not complain of pain on their being touched and handled. The general characteristic is that they are discrete, hard, and practically painless. Under these circumstances you would do well to bear in mind lymphadenoma, leucæmia, tubercle, syphilis, carcinoma, and beyond that there is another disease, a disease seen generally in younger persons, chronic enlargement of the glands, perhaps of the neck, perhaps of the axilla, usually diagnosed as lymphadenoma, though no increase in size occurs and yet they do not disappear, lasting sometimes for years. That form is a form of chronic glandular enlargement which to a certain extent resembles lymphadenoma, but it is non-progressive and it apparently does not do the patient any harm. There is no special name given to it, but it may be called chronic lymphadenitis. It is, as far as I have seen, a symmetrical enlargement, generally in the axillæ, and I have not seen it myself in the groin. The patient comes because he or she has lumps, and I warn you of it because you will probably see it diagnosed as lymphadenoma and a corresponding prognosis given. Perhaps it is lymphadenoma, but if it is it is not progressive. When a patient comes and you notice that on the left side of the neck above the clavicle there are enlarged glands, that should suggest to you the

possibility of visceral carcinoma. Of course the thoracic duct is in that region on the left hand side, and these glands on the left side are the first accessible glands liable to be affected in visceral carcinoma, as for instance in cancer of the uterus. In a doubtful chest case you may often make a correct diagnosis by feeling for these glands.

These lumps in this woman are not probably syphilitic, because syphilitic glands generally occur on both sides of the neck, and they are particularly liable to occur down the whole of the posterior border of the sterno-mastoid. They are probably not tubercular glands, because they are so very hard. On questioning the patient the points you learn are that the woman has been clearly "ill in herself," as the saying goes. She did not come because she had a lump in her neck, she came because she felt ill. The probability is that it is carcinomatous or lymphadenomatous. If I tell you that she has enlarged glands of the groin, and that, further, she has some hepatic enlargement, it is probably, as you will no doubt think, lymphadenoma.

Supposing that she has lymphadenoma, would you expect any pyrexia? There is a curious idea that patients with lymphadenoma do not have fever except when they have complications. Now, that is quite wrong; patients with lymphadenoma usually have fever, and the amount varies very much with the severity of the disease. The amount of fever they usually have is from 101° to 102° . I once had a patient with lymphadenoma with a temperature of 105° , without any so-called inflammatory complication.

The clinical varieties of lymphadenoma or Hodgkin's disease are hard and soft, that is what I was taught when I was a student; the hard variety was said to be a milder condition than the soft variety. Strictly speaking, you ought to recognise acute lymphadenoma and chronic lymphadenoma, the latter being by far the more common variety. I do not want to multiply and complicate matters, but it would be more satisfactory to recognise the following varieties. First, there is the ordinary form, which you may talk of as chronic lymphadenoma, in which the glands may be hard or the glands may be soft. Then there is acute lymphadenoma, of which I have had two cases, the whole duration of this disease is short, and my two cases were fatal within six

weeks of the onset. Ordinary lymphadenoma lasts much longer than that.

Another clinical classification is localised and generalised lymphadenoma. Local is what the surgeons generally see, and the general form is what the physicians see.

Etiology.—It is supposed to follow chronic irritation. Troussseau's view was that lymphadenoma arose from chronic irritation, and in the children of the poor you very often have it beginning in the neck from the frequency of pediculi. When it begins in other parts—in the axilla, for instance—it is thought very often to arise from a neglected sore. The modern idea of lymphadenoma is that it is a specific infection of the lymphatic system, produced by some micro-organism.

The disease is supposed to be infective because of the tendency to recurrence and to generalisation. Tubercle sometimes causes glandular enlargement of all the glands in the body, of course syphilis does too. There are thus two general infections with which to compare it. The way it occurs after acute diseases should also be remembered. I have seen it after scarlet fever—it might not be cause and effect, but it is interesting to bear in mind that in scarlet fever you have enlargement of the glands. The next point is the blood condition; there is considerable decrease in the amount of the red corpuscles, the white are very often increased. The cardinal point is the decrease in the red corpuscles, and you may have an actual increase in the white. The increase is in the form of lymphocytes more or less like the normal lymph corpuscles. In leucæmia the increase in so-called white corpuscles is very great. These are white corpuscles which have not got their counterpart in the normal economy except in the marrow of the bones. In leucæmia you have a totally abnormal form of white corpuscle present, you have what are called myelocytes. The point is that if in lymphadenoma the white corpuscles are increased, they are not the sort of corpuscles you find in leucæmia.

These patients come under observation usually for weakness or for the enlargement of their glands. Sometimes owing to a complication, e.g. pleurisy, they have very great weakness and the symptoms of anaemia, fever which we have men-

tioned, and they may have shivering fits. I once had a patient, a child 12 years of age, who was taken suddenly ill, with pain in the left side, with a temperature of 105° . She came in, and there was a mass near the spleen. On examination you could not get above the tumour, and it was very painful; there was nothing abnormal to show in the blood, and no great anaemia; she had slight general glandular enlargement, but it was so slight you could not lay much stress on it. The question arose was whether she had a rapid form of lymphadenoma or whether she had an abscess. I thought, on the whole, that she had an abscess, and got Mr. Pollard to make an incision, basing my diagnosis on the fact of the temperature and the patient being ill for so short a time, and the absence of any alteration in the blood. Well, she was operated on, and her spleen was found to be enlarged. She died six weeks after the commencement of the disease. She only had the traditional symptoms of the disease within the last fortnight of her life. That case simulated acute suppuration in every detail. She had pain in the hypochondrium, shivering fits, etc., and she had such slight glandular enlargement that it was nothing more than might have been quite normal. So you may confound the acute form of lymphadenoma with suppuration. Three years afterwards, in Ward IV., there was a man who had been taken ill suddenly with what was called pleurisy; it was a perfectly acute illness, and he came in here a month afterwards with the left side of the chest full of fluid; there was oedema of the chest and abdominal walls, and there were soft boggy swellings in the neck axillæ and groins, so soft as to be distinguished from the general oedema with difficulty. The patient was aspirated and the fluid was drawn off. It reaccumulated rapidly, and he died from asphyxia. That was a case which also ran its course in six weeks, and which simulated an acute disease. The ordinary cases of lymphadenoma have weakness, anaemia, a fair amount of fever, enough to give them shivering fits sometimes, and occasional haemorrhages from mucous surfaces. They have inflammatory complications, pleurisy and pneumonia, and they may die from asthenia as patients do in pernicious anaemia. More frequently, however, they die from inflammatory complications. Lymphadenoma, of all these anaemias, is the one

that is most liable to be complicated by acute inflammations, such as pleurisy; they frequently get peri-splenitis. They not uncommonly suffer from jaundice on account of the enlargement of glands in the portal fissure.

Prognosis.—In acute lymphadenoma you may mistake the boggy feeling in the axilla for oedema, whereas it is really due to swollen and very soft glands, and here the outlook is very grave. In the case of hard glands the disease runs a chronic course, probably a matter of one or two years; there is another thing which influences the prognosis, and that is the degree of leucocytosis, if there is considerable increase in white corpuscles, the prognosis is correspondingly bad.

Leuchæmia.

This child has a swollen abdomen and a pasty face. On palpating the abdomen you find that the spleen is enormously enlarged. You infer that this is splenic tumour from the position of the enlargement and the sharpness of its edge, the presence of a notch, and because the angle the mass forms with the costal margin is not acute. A renal tumour would give you a sharp or acute angle. The child has also an enlarged liver. Leuchæmia is more common in adults than in children. I have seen as many cases in women as in men. Gowers holds the view that leuchæmia is connected with ague, he says that 30% of these people have had ague. The Germans say that it is largely due to syphilis. It is not at all uncommon for leuchæmia to follow an injury. A sailor fell down a hatchway and broke several ribs in the lower part of the left chest, and shortly afterwards developed leuchæmia. It is then associated with ague and syphilis, and related to traumatism. Nobody ever sees an early case of leuchæmia. It is one of the most striking things in medicine that when you see these cases the spleen is already filling the abdomen, you never come across a slight case. It is clear, therefore, that there are two explanations, one is the patients are so well that they do not go to the doctor; the other one is that they do go to the doctor, but it is called something else. I personally believe that it is called something else, and that the splenic enlargement possibly precedes the blood change in some cases. It is not at all uncommon to see cases of splenic enlargement

with little or no blood change. Some of these are doubtless cases of splenic anaemia, but it is possible that some are early cases of leucæmia. If you do not make a routine of examining the blood you will miss the early cases. If you take diseases like pernicious anaemia lymphadenoma, Addison's disease, &c., all of you will probably see cases in which you are not quite sure, but you think a certain case may be an instance of one of these diseases, but you never see a doubtful case of leucæmia. As regards the initial symptoms they are usually vague. The ordinary adult with leucæmia comes to you complaining of a swelling of the abdomen. A woman I sent in to Dr. Bastian came complaining of various so-called neurotic symptoms and of a slight pain in her side. I thought she probably had hysterical tender points. I sent her round to be undressed for the purpose of making the routine examination, and found the spleen filling her abdomen. That is an illustration of the vague symptoms they complain of. They may come also for anaemic symptoms. They may come also for haemorrhages about the mouth. Sometimes urgent paroxysmal dyspnoea is a leading symptom. These dyspnæic attacks come on quite suddenly and are frequently very severe. The sailor who fell down the hatchway came for passing gravel. Patients with leucæmia often pass large quantities of uric acid. Swelling of the abdomen, asthmatic attacks, haemorrhages, attacks of renal colic, these are some of the symptoms that persons will complain of. It is usually looked upon as a rare disease, but you must remember that unless a routine examination of the blood is always made it may be very easily overlooked.

Purpura.

A patient was exhibited with subcutaneous haemorrhages. The haemorrhages were slightly raised above the surface, their outline was sharp, and the eruption was most abundant in the legs.

On suspecting purpuric spots, the legs, the trunk, and then the arms should be examined; it is a point to be remembered that the face is very rarely affected. Purpura may be primary or secondary. By a secondary purpura is meant a purpura accompanying some other morbid condition.

Secondary purpura is more common than

primary, and the simplest form is "mechanical" purpura, *i.e.* the purpura that arise from *morbus cordis*, or from thrombosis of veins.

In the so-called "mechanical purpura" the name is not meant to imply that the only factor in the case is the impediment to circulation, but rather that this variety of purpura is met with in conditions in which there is a mechanical obstruction to the venous circulation, but it is to be noted that this kind of purpura does not occur unless the patient is more or less cachectic. Occasionally, very severe purpura has resulted from venous thrombosis, and, some years ago, there was in Ward IV., a case that resembled the experimental purpura obtained by tying the femoral vein, and injecting thymus extract.

Another form of secondary purpura is "toxic" purpura, such, for instance, as the purpura produced by potassium iodide. Another salt of potash is said to cause purpura, *i.e.* saltpetre, and it is said that people who live on tinned and salted foods for a very long time may get purpura from the saltpetre used in preserving the provisions.

In cachectic conditions, more particularly in carcinoma, purpura occurs, and here its occurrence is of grave prognostic significance. The purpura occurring in renal disease is not a good clinical instance of cachectic purpura, since *morbus cordis* is often present in renal cases.

Purpura may occur in nervous diseases, purpuric spots may follow in tabes the lightning pains, and is sometimes called the neurotic purpura.

Clinically, the most common forms are potassium iodide eruption, cardiac purpura, and renal purpura; the other forms are not very often seen. This is only a clinical classification, and it is quite likely that the cachectic purpura may be a toxic purpura.

Purpura simplex and purpura haemorrhagica are the two principal forms of the primary variety, and the third form which is more rarely seen is purpura rheumatica, or peliosis rheumatica.

To distinguish between purpura simplex and purpura haemorrhagica the mucous membranes are appealed to, and if there is no bleeding from them it is a case of purpura simplex. In the patient exhibited, bleeding was demonstrated from the gums and the palate, and on being questioned the patient said he had been passing blood in his urine. The commonest places for bleeding are the mouth, nose, intestine, and urinary tract. In

the cases of hæmaturia several pints of blood may be lost, and the bleeding probably comes from the pelvis of the kidney, and not from the kidney itself. Purpura hæmorrhagica generally begins as purpura simplex, and that is why much caution is needed in cases of purpura when giving a prognosis, because it may or it may not become hæmorrhagic with a fatal termination.

Purpura rheumatica or peliosis rheumatica is a disease by itself, and that is why it is better to call it peliosis. It is something very different to a mere purpuric rash occurring in rheumatism, which is a comparatively common occurrence. In peliosis rheumatica there are initial joint-pains, then slight swelling of the joints with fever, e.g. 101° F., 102° F., rarely high, and a rash appears first round the affected joints, and subsequently more general. A fatal case exhibiting these symptoms occurred at University College Hospital in 1894. Another peculiarity concerning peliosis is that not only does the rash occur first of all round the joints, but it frequently begins as a raised papule, a papule which is quite easily felt, and then there occurs hæmorrhage into its centre, and as the hæmorrhage increases the papular character disappears, and the result is a petechial extravasation of varying size. Peliosis rheumatica is an affection untouched by salicylates, and further, cardiac complications do not occur, and these are some of the reasons why this disease is separated from ordinary rheumatic fever. In regard to the name peliosis rheumatica some clinicians term it an arthritic purpura.

In purpura hæmorrhagica bleeding from the intestine frequently occurs; the mechanism of these hæmorrhages is as follows: a hæmorrhage into the submucous tissue takes place and the mucous membrane sloughs, leaving an ulcer with a bleeding surface. One of the most formidable complications of peliosis rheumatica is extensive hæmorrhagic ulceration occurring in the intestine, and produced in this manner. These extensive and widespread ulcers cause not only hæmorrhage but also profuse diarrhoea, and it is scarcely necessary to point out that they are never observed in cases of rheumatic fever.

To recapitulate, in purpura simplex the hæmorrhages are limited to the subcutaneous tissue, in purpura hæmorrhagica they occur in the mucous membrane, e.g. the mouth, nose, and in the urinary tract, and also in the bowel, and the

patient may even expectorate blood from the lungs, a symptom more usually read about than actually observed. In peliosis rheumatica the eruption at first is localised round the inflamed joints and subsequently becomes generalised, and a condition may ensue in which the patient may succumb to severe hæmorrhagic diarrhoea.

There is another distinction between purpura simplex and purpura hæmorrhagica, besides the locality of the bleeding, and this difference is in regard to the size of the eruption; in purpura simplex the subcutaneous hæmorrhages remain small, but in purpura hæmorrhagica they may be very large, and if so sloughing of the skin is almost a certainty. One patient of mine in whom large vibices had occurred on the chest, as a result of peliosis, was subsequently demonstrated as a case of keloid, and the reason for the mistake was that practically the whole of the skin had sloughed away on the patient's chest, and the resulting huge scars were taken to be keloid. Another patient also suffering from peliosis had extensive sloughs in the perinæum and labia, encroaching on the vagina. The extensive ulcers produced in this way may add to the gravity of the case by becoming septic.

In purpura hæmorrhagica subserous bleeding takes place, sometimes petechial, at other times diffuse; the outer surface of the lungs may be covered, but it is very exceptional indeed to have any bleeding into healthy serous cavities. Cases have occurred in which, when the abdomen was opened (P.M.), the whole surface was uniformly black from subserous extravasation, with no blood at all in the peritoneum.

Cerebral hæmorrhage may occur in purpura hæmorrhagica, and bring it to a fatal termination.

Purpura is allied to hæmophilia and scurvy; it resembles hæmophilia, in the fact that in certain forms of purpura you have joint symptoms, and it resembles scurvy in the hæmorrhagic tendency.

Purpura hæmorrhagica may occasionally be secondary. I remember a case of a man with early and slight cancer of the pylorus, producing no obstruction and not ulcerated, who had suffered much privation before coming into the hospital. This man died from typical purpura hæmorrhagica. Cachexia may apparently bring about not only purpura simplex but also purpura hæmorrhagica.

Patients with purpura hæmorrhagica not unfre-

quently die. I have been on the look-out for the disease, and it is a good deal more fatal than you are apt to think, and I warn you about it, because one is apt to give a good prognosis.

Purpura hæmorrhagica cases may succumb to long-continued anæmia ; the amount of blood lost from a bad case is very great, considering that bleeding may occur at every mucous surface, with every act of micturition, with defæcation, with vomiting, from the nose and from the gums, thus much blood may be lost in a short time. They may also die from cerebral hæmorrhage, according to Bristowe. I have seen meningeal hæmorrhage in a fatal case of purpura hæmorrhagica, the whole brain was found covered by a thin layer of blood ; but Bristowe has described more than one case of death from cerebral hæmorrhage. Death may also occur from diarrhoea or from septic complications affecting the ulcerated areas left as a result of the extensive cutaneous hæmorrhages. Death then may occur from anæmia and its asthenia, cerebral hæmorrhage, long-protracted diarrhoea caused by the purpura hæmorrhagica and from intercurrent affections.

There is another interesting point ; patients with purpura may suddenly become blind, and that I have seen more than once. Sometimes it is due, perhaps, to retinal hæmorrhage, but I have seen one case where it was due to hæmorrhages into the sheathes of the optic nerves. This patient lost his sight suddenly, and no gross change was detected in the disc. This case was verified post-mortem.

Sometimes cases of purpura get progressively worse, notwithstanding the fact that all the external bleeding has ceased. A boy under my care was a case in point ; his external bleeding stopped, but his anæmia increased ; extensive visceral subserous hæmorrhages were found post mortem. It must be borne in mind that bleeding may occur under the peritoneum, but not into the cavity of the abdomen, quite sufficient to kill a patient and yet not capable of detection clinically.

In connection with purpura, erythema nodosum is interesting. Some regard erythema nodosum as one of the erythemata, others as a variety of purpura. In pure purpura there is a petechial hæmorrhage without any preceding eruption, and in pure erythema you have an eruption without any hæmorrhage, and midway between the two you

have erythema nodosum with its erythematous eruption and subsequent hæmorrhage.

It is not often in disease that three distinct grades can be so clearly demonstrated as is the case in passing from purpura to erythema nodosum and thence to erythema.

In treatment of purpura the two first things to consider are rest and food. The patient I show you here to-day got quite well when he was kept absolutely quiet ; but, as you see, on his walking about the purpura has returned, therefore absolute rest in bed is an essential point in the treatment. Further, these patients must have very good food, for though you cannot put your finger on the cause as you can in scurvy, still, there is little doubt but the purpura is due to dietetic defects. As regards drugs, iron and arsenic are the most useful. The student is supposed to say that hamamelis should be prescribed, but speaking from my own experience I should think that it might be claimed that its advantage at most is but doubtful.

A WEDNESDAY CONSULTATION AT ST. GEORGE'S HOSPITAL.

Displacement of Internal Semilunar Cartilage.

Mr. PICKERING PICK : This patient is a young woman aged 18. She has suffered from displacement of the internal semilunar cartilage, a condition formerly called internal derangement of the knee-joint. In March last she twisted her leg, and felt pain on the inner side of the knee and in the region of the knee-joint. The leg became fixed, and she could not completely extend it. This was followed by aching, and an attack of synovitis. On two subsequent occasions the same thing has happened, the third time fourteen days ago. There was then twisting of the leg, followed by acute pain, fixation of the joint, and swelling of the knee. This is a typical history of displacement of the semilunar cartilage. I recommend that in this case an operation should be performed. She is a housemaid and has to earn her own living, and any treatment to be of any use must be in accordance with the requirements of her position. Treat-

ment by clamps would certainly not be convenient. I therefore recommend that the joint should be opened, and the cartilage exposed and sutured. If the displacement had only occurred once I should have recommended keeping the limb in a fixed position so that adhesions might have formed, and then perhaps no operation would have been necessary. But considering that it has occurred three times, and that the patient is obliged to work for a living, and cannot afford to lie up for an indefinite period, I am of opinion that an operation is desirable.

Mr. HERBERT ALLINGHAM: The patient came under my care in the out-patient department. As the accident occurred only a short time before I saw her, I had her leg put up in plaster of Paris. After the plaster of Paris was taken off the accident occurred again. I then recommended massage to improve the muscles, because there was some wasting. That was done, but still on the slightest twist of the limb the cartilage gets pinched and she is unable to follow her work. On that account I sent her in to have the operation performed. As to what has exactly happened it is hard to say. The semilunar cartilage may be torn away from its posterior or from its anterior attachment, or the coronary ligaments may have been torn through or much stretched. I should advise, therefore, that the semilunar cartilage, if that be at fault, should be sutured to the head of the tibia or removed altogether.

Mr. MARMADUKE SHEILD: In these cases I personally in favour of first trying rest and the use of an apparatus. That would have been my treatment if the accident had occurred for the first time, but in this girl I should explore the joint. It is quite uncertain whether you will find conditions that you can do any good to, or whether a portion of the cartilage will be found detached. In this case careful exploration would be justifiable.

Mr. DENT: Considering the history in this case, the right thing to do is of course to explore the knee-joint. It is uncertain what you will find; it must not be taken too much for granted that you will find definite injury to the semilunar cartilage, but there is a strong probability that you will be able, by operative interference, to do some good in this case.

Mr. BENNETT: Understanding that this girl is prevented from following her occupation, I should explore the knee-joint to ascertain the state of the

internal semilunar cartilage. I should myself, if it is loosened, cut it away, for I believe that is a better plan than stitching, far more certain in its result, and free from any fear of recurrence of the displacement. I have seen two cases in which the displacement recurred after the stitching operation. By cutting away the piece of cartilage there can be no recurrence. I think that is the better plan if any loosening of the cartilage be found.

Mr. PICKERING PICK: I shall advise the girl to have the operation performed. I should like, however, to say something about this question of stitching as opposed to removing the cartilage altogether. You ought to be guided by the condition you find at the time of operation. The last case I operated on, an officer from India, who was a great football player, and had displaced one of the semilunar cartilages on many occasions, I found that the anterior attachment was torn away. I sutured that down, and the patient is now perfectly recovered, and no kind of recurrence has taken place as yet. On other occasions, under different conditions, I think the cartilages should be removed, but I would not lay down any rule, and the only way is to explore the joint and see what the conditions are, and then decide on the treatment.

Double Congenital Hernia in an Infant.

MR. DENT: This is a case of a double scrotal hernia in an infant, and there is also, as is usual in these cases, an affection of the prepuce. This infant has worn a double truss pressing on various parts of its abdomen, but it has never had a truss which has succeeded in keeping either of the herniae back, and I do not think there is any chance of its getting any. The question is whether the child should be operated on. With regard to operating on very young children, I think they run rather more risk than at a later period. The child in this particular case will go on straining, the herniae cannot be kept back, and there will, I think, be no chance of any natural tendency to cure. I propose, therefore, an operation. I do not care to operate on both herniae at the same time. The position of the parents is such that they cannot afford to provide suitable attention and the necessary trusses.

MR. HERBERT ALLINGHAM: I agree that it is a case for operation; the child's parents are poor, and they

cannot afford to pay for trusses nor can they give proper attention to the case. If they were in better circumstances and well-to-do, with a great deal of care and trouble these herniae might be kept up, and so they might cure themselves. I should operate first on one side, and leave the other for another operation ; at the same time I should circumcise the child.

Mr. MARMADUKE SHEILD : I should do both the herniae at the same operation ; the principal risk is that of the wound getting septic. I do a great many of these operations, and I find that the main difficulty in young children is to keep the wounds from becoming septic. I prefer waiting till the children are four or five years of age. There is a difference of opinion as to the age at which these operations should be done ; I believe the younger the child is the better it does, provided you can keep the wound aseptic, but that of course, as I have said, is a very difficult matter in young children.

Mr. BENNETT : I think this is a case in which a radical cure is called for. My experience in these cases does not lead me to notice the same difficulty as Mr. Marmaduke Sheild. I allow that there is very rarely a liability in three or four days after the operation to get a little superficial suppuration about the wound. That does not affect, however, the progress of the case. The peritoneum and the under parts are completely sealed by that time, and I have never had any anxiety in this respect in a large series of operations. I should not do the operation on both sides at the same time ; I used to do so, but I found that when I dealt with the two sides together, curiously enough an umbilical hernia was prone to follow. It seems as if on pushing back the hernial contents on the two sides at the same operation some extra intra-abdominal pressure were made, which produced the umbilical hernia. This resulting umbilical hernia occurred in six cases of mine one after the other, and I then gave up doing the two operations at one time, and began doing one side first, then dealing with the other side later on. I found that plan gave more satisfactory results.

Mr. PICKERING PICK : I know that there is a great difference of opinion as to the propriety of operating on these cases. There are two objections, which are usually urged ; the first is that the wounds

are likely to become septic. I always teach that that is a question of nursing. My experience has not been that of Mr. Marmaduke Sheild. I have done many at the Victoria Hospital for Children, and I should certainly blame the nurse if the wound got septic. The second objection is an important one. Surgeons urge that these cases can be cured by wearing a truss ; in this case the child has worn a truss, and it has not succeeded in curing the hernia. But it is an undoubted fact that many cases appear to get well after a time by wearing trusses, but I am not sure whether they are permanently cured. In the congenital form the funicular process gets narrowed from wearing a truss, but is not completely obliterated, and later on in life the hernia may recur. I have known many cases of adults coming for treatment of hernia, who have said that they had a hernia when they were children, but that it was cured and had now recurred. Besides, the application of trusses in young infants is a very difficult matter ; it is constantly slipping, and requires replacing ; it is frequently soiled, and soon gets spoilt and requires renewal, and is a constant expense to the parents. I am, therefore, strongly in favour of operating in young children.

Mr. DENT : There is one point to be noted in regard to keeping these wounds from becoming septic, and that is to abstain from using too great a quantity of dressing, which is apt to become saturated with urine, and becomes rather a source of infection than of protection.

Hip-joint Disease.

Mr. MARMADUKE SHEILD : This is a patient of 14 years of age. He was quite well up to five weeks ago, when there was suddenly an acute pain and swelling in his hip ; no history of injury can be procured. He suffered from severe delirium and great headaches, but, however, he has got quite rid of these symptoms. The idea at first entertained was that it might be tubercular meningitis. There is a swelling in front of Scarpa's triangle, and the question arises as to what is to be done. The disease probably commenced as a tubercular osteitis. I think it is a bone case to begin with ; whether the pelvis is affected or not is another question. I think very likely it is, but I do not know for certain, so I thought it would be right to make an exploratory incision, excise the head of the femur, scrape out the acetabulum, and drain it posteriorly. The question lies between that operation and a Furneaux Jordan's amputation. Seeing that it is a case of considerable danger, I should like to have the opinion of my colleagues.

[August 5, 1896.]

A formal excision of the hip is one alternative, and the other that I think might be indicated is whether I should perform a Furneaux Jordan's amputation.

Mr. HERBERT ALLINGHAM: I should only open the abscess.

Mr. MARMADUKE SHEILD: Supposing you find necrosis there?

Mr. HERBERT ALLINGHAM: I should then simply remove the diseased portions. Considering the head symptoms the patient has had, I should wait and see how the case goes on, merely opening the abscess.

Mr. TURNER: I think, too, that on the whole the abscess might be opened and dealt with first of all. I think with Mr. Allingham that one should postpone anything more. I should not at present amputate.

Mr. DENT: I think the abscess should be opened, and I should open it so as to get to the joint. I am inclined to believe that the mischief began in the head of the bone. A number of cases have occurred here in which mischief of the hip joint was associated with an early development of meningitis. The patient has had symptoms of trouble of that sort. At the time of opening the abscess I should explore to see if the head of the femur or upper part of the bone was involved, and in that case should remove it. For my own part I think that I have seen the best results after excision when the disease is central, and when the head of the bone with the contained sequestrum is removed early. It is noticeable here that there is a fair amount of movement of the hip-joint; without flexing the leg on the pelvis, you get a slight rotation of the head of the bone of the pelvis without pain.

Mr. BENNETT: A free incision and thorough drainage is the treatment I advise; but I should be quite sure that I got thoroughly into the diseased parts, because I agree with Mr. Dent in thinking that there is probably loose bone present. Considering the character of the case, I should deal with it as an abscess in which I expected to find loose bone requiring removal.

Mr. PICKERING PICK: It seems that this is a case of septic inflammation of the bone, most probably in the growing tissue at the extremity of the shaft of the bone on the under surface of the epiphyseal cartilage. I believe it partakes very much of the character of those cases which used to be called acute diffused periostitis, or by the Germans osteo-myelitis, and now known as acute necrosis. Mr. Bennett has summed up the treatment: free incision and drainage. But this free incision must be into the centre of the bone, and what I should do would be to make an incision in front as for excision of the hip. In this way I should expose the neck of the femur, and if I found it thickened I should freely open it and

scrape away all the inflamed tissue, and if the joint were implicated I should incise the capsule and drain. I should not, however, excise the head of the bone. There is a slight movement in the joint, and although it may be full of pus I do not think the head of the bone is involved in the disease. I think more probably the disease is in the neck of the bone. Excision of the hip-joint is not a satisfactory operation if done in the acute stage; and in this case it may not be necessary, if only you can find the focus of inflammation and get rid of all the infective material. If later on it must be done, it will be done under more favourable circumstances after the more acute symptoms have passed off.

Sarcoma of the Fibula.

Mr. MARMADUKE SHEILD: This is a child of 5. Some years ago she had a tumour in the left leg, which was removed once, or somebody is said to have tried to remove it, but since then it has been growing, and there is a large tumour here on the leg apparently connected with the periosteum of the fibula. There are no pelvic deposits and no glands, neither are there any signs in the lungs or viscera. I fear myself that it is a sarcoma. Of course, the question comes in of congenital tumour, but I think that is quite out of court. It is the youngest case I have seen of sarcoma of the bone; I have seen it in the eye and in the kidney in younger cases, but I have never seen it in connection with the bone in a younger patient. I have seen the reports of cases, but anyway it is rare. I do not think it is possible to remove that tumour locally. There is no history and no signs of congenital syphilis about the child. I suppose that there can be no question that the right treatment is amputation of the limb at the lower third of the thigh? The prognosis is unfavourable, and recurrence will probably occur in this case.

Mr. HERBERT ALLINGHAM: If an amputation is performed it should be done in the lower third of the thigh.

Mr. TURNER: I think an amputation is the right thing to be done. It would be interesting if Mr. Marmaduke Sheild could procure details of what was done when the child was supposed to have been operated on on the appearance of the growth three years ago. It is the fashion now to remove these tumours locally, leaving just the shell.

MR. DENT: It is just possible that if Mr. Sheild were to run through the literature of the subject he might find this case reported as a case of recovery. I should amputate as suggested.

MR. BENNETT: The only treatment is amputation, and the prognosis must be very unfavourable.

MR. PICKERING PICK: I quite agree with the treatment proposed and the unfavourable nature of the prognosis.

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A CLINICAL LECTURE ON PNEUMONIAS IN CHILDREN AND THEIR SEQUELÆ.

Delivered at the Victoria Hospital for Children, Tite Street, Chelsea,

BY

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PROBABLY few medical terms are used more frequently or more glibly than is the term "pneumonia," to signify any inflammation of the lungs. Few words, too, cover a larger number of conditions; for pneumonia is not one disease, but many diseases, with many different causes; nor is this surprising when we consider, firstly, that all the germs in the air have free access to the lungs; secondly, that all the blood in the body is constantly being passed through the lungs, but at different rates and varying much in composition in different diseased conditions; and, thirdly, that whilst the lungs are never at rest, their range of movement varies greatly in varying conditions of disease. In adults, the more we investigate pneumonia the more varieties we discover: acute and chronic, active and passive, primary and secondary, septic and infectious, &c.; and so also in children we find the same varieties, but with certain well-marked differences from the adult forms in relative frequency, in symptoms, in course, and in prognosis.

Croupous Pneumonia. — In adult life, when we speak simply of pneumonia, we mean usually the croupous or fibrinous form, which from its sudden onset, its rapid tempestuous course, its generally marked physical signs, and its definite duration, attracts special attention. Hence we naturally inquire first of all whether this form occurs in children, and if so with what fre-

quency and with what special features? Turning first to experience in the post-mortem room, we find that croupous pneumonia, as seen in adults, is distinctly rare in children. True it is that we find many cases of "lobar" consolidation, that is to say, the pneumonia is geographically "lobar," or may even involve the whole lung; but pathologically it is not true croupous pneumonia, for in these "lobar" cases in children we do not find the dry, uniformly granular appearance so characteristic of croupous pneumonia, but the lung has a mottled look, it often contains a little air intermixed with the consolidated patches, and the bronchi are found on pressure to be full of thick pus; whilst, if we turn to the course and symptoms of these cases during life, we find that they are different from those of true croupous pneumonia. Of 100 consecutive cases of pneumonia in children of which I have post-mortem records, only 11 could possibly be considered examples of the croupous variety, and these were all in children under two years of age, and nearly all either secondary to measles or else to some marasmic or septic condition. In several of them, although the post-mortem appearances suggested the croupous form, the course and especially the temperature indicated a departure from the normal type. Furthermore, out of some 500 post-mortems on children, which I have performed at this Hospital, amongst those over two years of age only two could be said to have suffered from acute croupous pneumonia, and in neither were the symptoms during life very typical.

Very different is the experience in the out-patient room and in the wards; in both we see a large number of children with all the signs and symptoms of ordinary croupous pneumonia. Amongst the out-patients a not uncommon history is that for a few days the child was so ill that the mother was afraid to take him out, but as he seemed a little better he is brought to the hospital; on examination, typical physical signs of pneumonic consolidation are often found, but the child no longer seems ill, and has a perfectly normal tem-

perature; evidently he has passed through an attack of pneumonia, and the crisis has already occurred: the subsequent progress of the case confirms the diagnosis, for recovery is usually rapid and complete. The disease occurs in children at all ages, but is not common in those under two years. We conclude, therefore, that clinically croupous pneumonia in children, excepting infants, is a common disease, whilst in the post-mortem room it is distinctly rare; and the inference is probably a legitimate one, that it is rarely fatal.

Now what is the course of croupous pneumonia in children? How does it differ—besides in fatality—from the adult form? What troubles may it give rise to in diagnosis? What especially are the points of difference from bronchopneumonia? The onset is invariably sudden, and most commonly, in my experience, with vomiting; then follow all the symptoms of severe illness and high fever, with in addition notably rapid breathing; there is usually no diarrhoea (the significance of this will be seen presently). The temperature is continuously high, not necessarily or even usually keeping constantly at the same level, but with moderate fluctuations, always remaining considerably above the normal, with sometimes more marked remissions towards the close. From the fifth to the tenth day the disease ends with a sudden crisis, the temperature often falling six to eight degrees in twelve hours: there may possibly be a post-crisial rise on the following day, followed by complete and permanent subsidence of the fever. Recovery is usually rapid.

What are the physical signs during this time? Frequently no abnormal signs whatever can be detected until quite late, sometimes not before the crisis. They seem often to be later in developing in children than in adults, perhaps because in the former the consolidation frequently commences about the root of the lung, at a part over which distant bronchial breathing is of doubtful significance. I doubt, however, if any cases occur in which some evidence of consolidation may not be detected during the course of the disease, although during the first few days we must often be prepared to make a provisional diagnosis from the symptoms only.

Diagnosis.—1. In a child, high fever of sudden onset, with vomiting, and at first no physical signs, suggest, of course, one of the acute specifics, and

we can only exclude them with certainty by waiting, and noting the non-appearance of the rash. Fortunately, scarlet fever, the most likely one of all, can with certainty be excluded on the second or third day by the absence of rash and sore throat. Of course, day by day the chest should be examined in the hope of finding slight dulness, or some distant tubular breathing which would indicate consolidation of the lung.

2. In some of these cases cerebral symptoms markedly predominate, probably not in the majority, but when such do occur, they attract special attention; convulsions, vomiting, squint, head retraction, may all be present, and strongly suggest brain disease, especially meningitis. There is no expectoration, and therefore no rusty sputum to help us in the diagnosis; but, of course, the physical signs should be always carefully watched in a doubtful case; and above all, the rapid breathing, with working of the alæ nasi, should be noted. Unless thought of, this is more easily missed in children than in adults, because children, especially when feverish, always breathe somewhat rapidly, and therefore the marked panting dyspnoea of pneumonia may not attract attention, unless the importance of observing it be borne in mind. Examination of the urine may also help, as in adults; chlorides, in cases of pneumonia, are often absent, or at any rate, greatly diminished, whereas in meningitis they may be present in normal quantities.

3. An acute consolidation of the apex may, of course, be tubercular; and often we cannot decide, until the crisis occurs, whether we have to deal with acute phthisis or with pneumonia, but it has happened to me that after the crisis, on seeing a child for the first time, I have mistaken a resolving pneumonia for a rapid tubercular softening. A child is brought up to the out-patient room with signs of rapid breaking down at one apex; there is a history of previous illness, but its duration and symptoms cannot be definitely ascertained, and it is very easy to regard the case as one of rapid softening of the lung, and to give a prognosis accordingly; whereas, a fortnight later, it may be found that the abnormal physical signs have entirely cleared up. But alike in regard to meningitis and pulmonary tuberculosis, an adequate recognition of the possibility of mistaking pneumonia for either of them, is the best safeguard for avoiding the error. The pitfalls into which we fall are

those of which we are not aware, or not watching for.

Sequel.—I have said that recovery is usually rapid; in fact, the only complication at all frequent is empyema. Pneumonia is commonly accompanied by a certain amount of pleurisy, and in children inflammatory exudations are notoriously richly cellular; the result is that the pneumonic lung frequently becomes coated with a very thick layer of lymph, and sometimes this lymph, instead of being absorbed, slowly softens, and then an empyema results. In any case in which, after acute pneumonia, the temperature becomes hectic, the child wastes, and dulness remains, the chest should always be explored at frequent intervals with an exploring syringe and a needle of large calibre, for the thick, half broken-down lymph would not pass at all through a small needle. The auscultatory signs in these cases are often misleading.

Prognosis.—I have already said that, except in children weakened by severe rickets, marasmus, or acute specific disease, death from croupous pneumonia is very rare, and after all this is only what we should expect from the prognosis of acute pneumonia in adults, for we know that its fatality increases rapidly with age. In previously healthy young adults the death-rate is small, in children the same process is continued until it becomes almost infinitesimal.

Treatment.—Obviously from the foregoing, little special treatment is required, stimulants are rarely necessary, and antipyretics still less frequently desirable. Local applications of ice may possibly be beneficial at times, and in complicated cases, but can seldom be called for in a disease in which there is such a great tendency towards spontaneous recovery. Often I do not even admit these cases to the hospital, especially if there be any suspicion of specific fever when they are first seen; by the second visit, three or four days later, they have usually had their crisis, and convalescence has commenced.

We now turn to *Broncho-pneumonia*. This is the commonest form of pneumonia in infants, and certainly the variety which is usually found post-mortem. In 100 autopsies on children dying from pneumonia, 81 were under two years of age, and the great majority had broncho-pneumonia. In children it shows enormous variations alike in

its onset, course, symptoms, and physical signs. The onset is generally gradual and may be preceded by laryngitis and bronchitis, but sometimes it is distinctly sudden. The symptoms are those of a severe illness, with a marked look of illness, the child being apathetic, indifferent to toys, and entirely occupied in breathing. Of course there is cough, usually frequent and severe, but the most marked symptom is the dyspnoea, the alæ nasi work, and the breathing—especially expiration—has a peculiarly grunting character, which is very typical. Diarrhoea is a common symptom; for in children the bronchial and gastro-intestinal mucous membranes are closely allied pathologically, and both bronchitis and broncho-pneumonia are usually attended with diarrhoea, often very troublesome, seriously increasing the gravity of the case, and intractable to treatment. I have already drawn attention to the fact that in true croupous pneumonia diarrhea is not common. The temperature is markedly irregular in type, varying in twenty-four hours often between 98° and 105° ; note especially that it is not always highest in the worst cases. The duration of the fever is most uncertain,—it may be only a few days, it may be some weeks; the fall is gradual; in all these respects the temperature chart of a case of broncho-pneumonia presents very typical distinctions from one of croupous pneumonia. (Compare Charts 1 and 2.)

The physical signs depend on the extent of the consolidation and especially on its uniformity; small solid areas, even though very numerous, may not give rise to any special signs, and it should be remembered that in infants severe bronchitis is nearly always attended with some broncho-pneumonia. In some cases the physical signs are merely those of bronchitis, with much recession, and in places sharp, somewhat consonating râles, more distinct than those of simple bronchitis. In more typical cases we get patches of dulness, bronchial breathing, consonating râles, and increased cry resonance; whilst in the extreme cases in which a whole lobe is involved, the signs are those of croupous pneumonia, but usually with more abundant moist sounds.

We meet, moreover, with a considerable number of indeterminate or mixed cases of pneumonia in children—insidious forms, occurring especially in marasmic babies, in which we get a mixture of

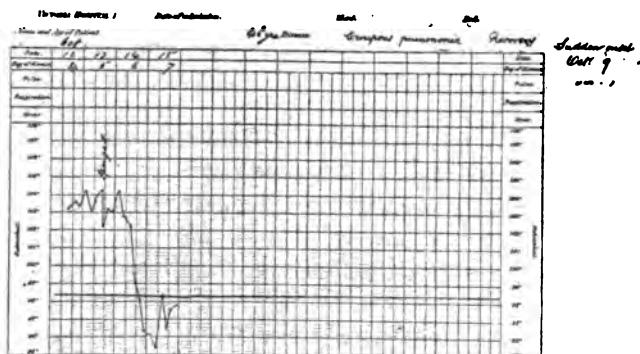


CHART No. 1. Croupous Pneumonia.

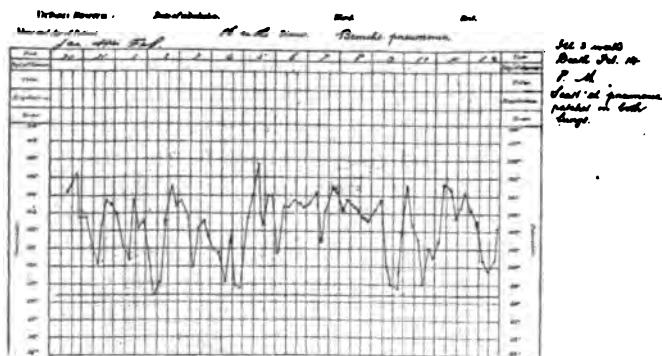


CHART No. 2. Broncho-pneumonia.

collapse, hypostatic congestion and inflammation, often coming on with little or no fever and no urgent symptoms. These cases are similar to many met with in adults, especially in the later stages of severe illness or asthenic conditions, and often admit of no definite classification.

Furthermore, we must allow that there are many borderland cases between croupous and broncho-pneumonia, in which it is impossible to say during life which condition is present, and sometimes very difficult to distinguish after death; in fact, there is no doubt that both varieties are often combined in children,—croupous or fibrinous pneumonia in one lung or one part of a lung, and broncho-pneumonic patches in other parts. Clinically, however, one may say that the more irregular the temperature, and the more prolonged the course of the disease, the more broncho-pneumonic is it in type, and the more serious is the prognosis.

The majority of cases of broncho-

pneumonia terminate in a few weeks in death or resolution, but a certain number do not clear up, especially those which occur after measles and whooping-cough, or in children previously unhealthy or rickety. Dulness, consonating râles and an irregular temperature persist, and in such cases the diagnosis from tubercular mischief becomes exceedingly difficult; in fact, in many instances of broncho-pneumonia, even in the earlier stages, the resemblance between these two diseases is remarkably close. On looking over the records of lung cases, which I have sent into the hospital from my out-patients, I find every year that I have admitted some cases as tubercular which have turned out to be pneumonic, and others which I have diagnosed as pneumonic have proved themselves to be tubercular. In the prolonged cases of broncho-pneumonia the diagnosis sometimes becomes excessively difficult, if not impossible; the duration gives no help, the symptoms are just the same in both, the physical signs are often identical alike in character and in distribution through the lungs, whilst the temperature chart of a broncho-pneumonic case may be exactly similar to that of a case in the hospital at the same time which turns out to be one of disseminated tuberculosis. (Com-

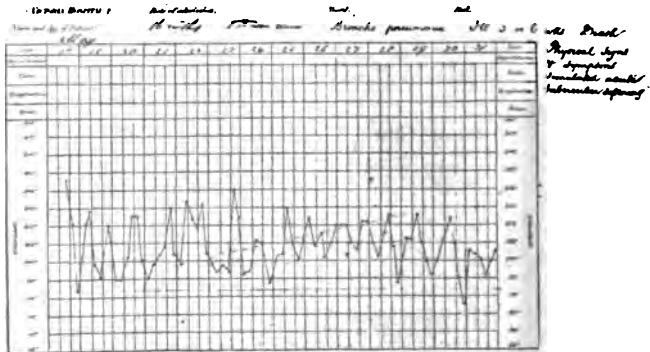


CHART No. 3. Broncho-pneumonia.

Physical signs and symptoms simulated acute tubercular softening.

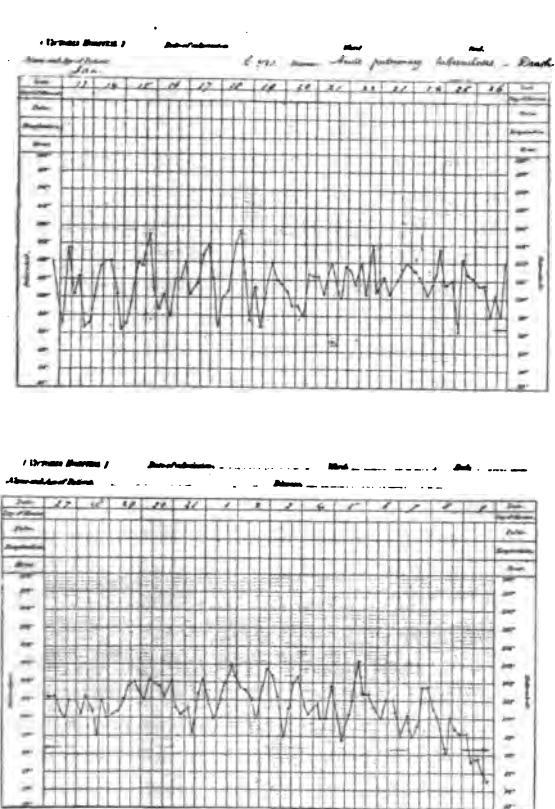


CHART 4. Acute Pulmonary Tuberculosis.

pare Charts 3 and 4.) The only points I can mention which are at all likely to help are these:—First, that in phthisis, the râles may have a more distinctly liquid character; and secondly, that in older children, a doubtful case is more likely to be tubercular, whilst pneumonia is commoner in infants.

Now, if the diagnosis be so difficult, are the results of a possible error serious? The mistaking of phthisis for broncho-pneumonia cannot lead to any grave results as regards treatment, although the medical man may possibly suffer somewhat in reputation should he have been led to hold out a probability of recovery. More serious is the result of mistaking a broncho-pneumonia for tubercular mischief. There is a danger of regarding the case as hopeless, and therefore of becoming somewhat negligent in treatment, a neglect which is probably communicated with increased intensity to the nurses and friends. Occasionally, on finding at a post-mortem that a case which I had thought to be one of pulmonary tuberculosis was really

broncho-pneumonia, it has occurred to me as a possibility, that had a more hopeful view been taken during life, and tonic and stimulant treatment and food more persistently urged, the fatal result might have been averted.

Another possible error in dealing with these cases is that of overlooking an empyema. As a matter of fact, empyema does occasionally—though not often—supervene upon a broncho-pneumonia, and the only way to avoid the danger of missing it is, in any case in which the temperature and physical signs persist, to frequently explore any dull area with a syringe and large needle.

A broncho-pneumonia, however prolonged, should never be despaired of, for the wonderful recuperative power of children is seldom more markedly shown than in cases of this description. There has recently been in the wards a child, who for eight or nine weeks was extremely ill, had a most irregular temperature, and persistent signs of consolidation at the right base. Yet, after that time, recovery has occurred. The child is now comparatively stout, almost entirely free from symptoms, and the only physical signs left are very slightly deficient resonance and a few dryish crackles at the right base. Records of such cases might be multiplied from our experience here.

Many of these children, of course, do eventually die from exhaustion, or from increasing lung trouble, and we find at the autopsy that large areas of lung are solid, present a mottled appearance, and are usually still soft in consistence, though possibly firmer than at an earlier stage. But what we should especially note is that the bronchial tubes show slight but distinct dilatation throughout the consolidated areas; and this dilatation, if looked for, may even be noticed in a catarrhal pneumonia that has lasted only a very few weeks.

A certain number of cases, however, still remain, which neither die nor clear up, but drift on not merely for weeks, but for months or even years. Over a small area of lung, or sometimes over a large part of the chest, dulness and consonating râles persist, and presently more distinct signs of excavation—cavernous breathing, and gurgling râles—show themselves. In such a case the physical signs strongly suggest a rapid disintegration of the lung; and if a child be seen for the first time when in this condition, the tendency to

diagnose tubercular mischief is almost irresistible, and even when it has been watched for a considerable period, it is difficult often to avoid this fallacy in diagnosis. But disseminated tubercular mischief in a child's lungs is a condition which causes severe symptoms, a markedly hectic temperature, and a rapidly fatal issue in a few weeks, whereas these chronic broncho-pneumonic cases live for months or even for years. They often have for long periods of time a normal temperature, and sometimes even gain weight—conditions which are quite incompatible with pulmonary tuberculosis. The clue to this condition is furnished by the slight dilatation of the bronchial tubes which I referred to just now as taking place at a comparatively early stage of broncho-pneumonia; the fact is that when the case becomes chronic, definite bronchiectatic changes occur, the cough becomes more and more paroxysmal, the dyspnoea slowly increases, eventually cyanosis and clubbing of the fingers become well marked, and these children usually die at last either from simple exhaustion or from an intercurrent attack of bronchitis. At the post-mortem we find well-marked cylindrical dilatation of bronchi, with more or less cirrhosis of the intervening lung tissue.

I wish to lay special stress on these cases, because I believe they are frequently overlooked in children, being wrongly diagnosed as tubercular. Many examples could be given from our experience here. I may mention one; a rickety child, *æt. 2* years, was admitted to the hospital with general bronchitis and signs of consolidation over the right base. The physical signs remained much the same, but over the area of consolidation the breath sounds gradually became more tubular, and the râles more numerous and consonating, but for all that the general condition improved, and the cough diminished. Three months later the right side was dull all over, with sharp clicking and gurgling râles everywhere, and more cavernous sounds at the base; there were similar but less marked signs over the left lung. The cough was spasmodic, and the breathing rapid. The early diagnosis had been one of tuberculosis, but this now had to be abandoned, and in spite of the signs of extensive and severe lung disease, the child for the next four months continued to improve, and gained three pounds in weight. After this the patient slowly got worse, with increasing

cyanosis, and died from exhaustion, after being nearly eight months in hospital. There was never any fever of the breath. The temperature was irregular for the first two weeks; afterwards it hardly ever rose above normal, and during the last few weeks of life was persistently subnormal. Such a temperature chart would have been impossible had the extensive physical signs present been due to tuberculosis. At the post-mortem, the bronchial tubes throughout the right lung, especially in the lower lobe, showed everywhere marked cylindrical dilatation, and the lung substance was much tougher than natural; similar, but slighter changes were present in the left lung. I have met with several similar instances.

In these cases the diagnosis must turn upon signs of lung excavation, combined with symptoms not sufficiently urgent, a temperature rarely sufficiently raised, and a course far too chronic for tubercular mischief, as it occurs in the lungs of children; together with markedly paroxysmal cough, sometimes causing sickness. Fœtor is frequently absent altogether, or, at any rate, for a very long time. In the case above narrated it was never present, and I believe we are fully justified in diagnosing cylindrical bronchiectasis in children in its absence. Another possible error in the diagnosis of these cases is to mistake them for whooping-cough. The history should prevent this; but in hospital I generally find that nurses regard them very dubiously, so closely do the paroxysmal attacks of cough simulate those of pertussis.

Treatment.—There is no need for me to enter upon the ordinary treatment of broncho-pneumonia in children, but I may say a few words about the method of dealing with these prolonged cases, to which I have made special reference. I believe the most important element in treatment is to put the children under the most healthy conditions possible, in the hope that as their vitality improves the lung condition may improve also; hence I am no believer in keeping them in a steam tent. In the early stage this may be useful, but later on, by excluding light and air, I believe it only retards recovery. The same thing holds good with regard to expectorants and all depressant drugs. When one sees these cases at the post-mortem it is impossible to conceive that an expectorant could

exert any influence on the lung condition, and I believe that tonics, cod-liver oil, maltine and iron, combined often with stimulants, as much suitable food, and as pure air as possible, are the most important factors in promoting recovery, and preventing them from drifting into incurable bronchiectasis.

NOTES AT THE INTERNATIONAL CONGRESS OF DERMATOLOGY.

LONDON, AUGUST, 1896.

Syphilitic Re-infection.

MR. ALFRED COOPER and Mr. EDWARD COTTERELL (London) contributed a paper to demonstrate that one attack of syphilis, although generally conferring lifelong immunity, does not always do so, and that re-infection of syphilis, although rare, is certainly possible; that great care must be exercised before accepting many of the cases published as second attacks of syphilis; that a proper and judicious course of mercury will cure syphilis, as demonstrated by re-infection of the disease being possible; and that hereditary syphilis as a rule confers immunity to the individual, but that there are exceptions to this rule.

Dr. HENRY FITZGIBBON (Dublin) considered that syphilis was a specific fever of the same class as the other major exanthemata. If uncomplicated by pre-existing constitutional cachexia, or co-existing septic influence, it runs a definite course, by which it exhausts in the system of its recipient the elements upon which its virus can feed. Thus, like variola, vaccinia, &c., the first attack is followed by a period during which the same individual is insusceptible of re-infection. The effects of syphilitic infection are no more necessarily life-long than those of any other zymotic eruptive fever, but that the process by which it is eliminated is more tedious and is liable to interruptions and complications which are not common to it with the other exanthemata. There is abundance of evidence that by far the greater number of persons who contract syphilis recover completely from it, and there is also indisputable proof that after the lapse of a period of over five

years not only has the disease disappeared from the system, but that even the protective influence of it may die out, and the elements which it had exhausted be re-established. Too much importance has been attached to the question of the possibility of second infection with syphilis, as the only reliable proof of complete recovery from the disease. The experience of all those who have much knowledge of the treatment of the disease in the present day is, that with few exceptions, complete recovery takes place within three years from the date of infection, and that the subject develops no subsequent evidence of the disease either in his own person or by transmission to his offspring. It may happen, as it does in smallpox or vaccinia, that the protective influence remains, but this is no evidence that there is any syphilitic taint left. It would be equally rational to assert that a person was still suffering from smallpox, vaccinia, scarlatina, or typhus fever, because they were still under the protective effect of one of these exanthems and insusceptible of re-infection. From reported cases it would appear that when second infection with syphilis takes place, the disease is more likely to be of an aggravated type than in first attacks. From this he concluded that re-infection was more apt to be communicated by contact with the impure or septic source of infection than from pure and less virulent syphilitic virus. In illustration of this, he referred to a case he had himself reported, and also to those reported by R. W. Taylor of New York, two of which "ended quite promptly in death." Such a consequence was more suggestive of acute sepsis than re-infection with purely syphilitic virus.

The Duration of the Period of Contagion of Syphilis.

Mr. JONATHAN HUTCHINSON (London) assumed that it was generally acknowledged that during the existence of the primary and secondary phenomena, the blood and all inflammatory secretions contained the specific virus of syphilis and were capable of conveying it. The question was the period at which, as a rule, these fluids cease to contain the virus and to be capable of conveying the disease. It was also assumed as an acknowledged fact that parents (whether father or mother) having suffered from syphilis might convey the disease to offspring, and the question was

raised as to how long this possibility might last and when it usually ended. It would be freely admitted that exceptional cases of long-continued possibility of contagion might occur in reference to the communication of syphilis, both by direct contagion and by the contamination of the foetus. He asserted, however, that, as a rule, it was not much longer than a year and but very rarely more than two years. He further maintained that it was probable that the vigour of the contagious element, or perhaps its abundance, diminished in ratio with the duration of the disease, and that contagion was far less certain during the later stages of the secondary period than during the earlier ones. The great rarity of cases in which during the tertiary period (after the end of the second year) suspicion arose as to either contagion or hereditary transmission was strongly insisted on. Cases were cited to illustrate (1) Exceptionally long periods of survival of contagious properties; (2) Exceptionally long periods in reference to hereditary transmission; (3) Manifestations of great potency of the virus (as in series of vaccino-syphilis cases) in reference to the stage of the disease; (4) The escape of those who were known to have been exposed to imminent risk, as bearing upon the doctrine of particulate germs.

Professor CAMPANA (Rome) considered that the period of the duration of the contagiousness of syphilis could not be determined with certainty. As a matter of fact, it existed as long as the anatomical manifestations of the syphilitic disease, which had no tendency to undergo caseation, and which were characterized by the manifestations of the so-called virulent secondary period of syphilis. In fact, it lasted as long as there were manifestations capable of giving rise to the anatomical phenomena of inflammation without subsequent caseation. Modern theories on the syphilitic process did not exclude the possibility of gummatæ occurring in its early stage or inflammatory period, yielding contagious, parenchymatous juices. Practically, the infective period of syphilis lasted for the whole time during which the disease was hereditarily transmissible. This period was mainly dependent on anti-syphilitic treatment, which, if carried out properly, justified the hope that after three years the disease itself and its results might disappear.

Professor LASSAR (Berlin) considered that the infective power of syphilis decreased with the length

of duration of the disease, but might persist as long as symptoms manifested themselves. Literature, practical experience, and experiment gave negative results, but in principle they did not exclude the possibility of contagion from later syphilitic manifestations. Infection from later manifestations was scarcely ever observed, owing probably chiefly to the localisation of the eruptions; but (as in leprosy and tuberculosis) other conditions must be taken into consideration which either prevented or permitted contagion. Justifiable doubts were, until recently, entertained as to the contagiousness of condylomatous eruptions, which was now fully admitted. In the same way a similar solution of the question of the contagiousness of later syphilitic manifestations might soon be forthcoming.

Dr. H. FEULARD (Paris) considered that the length of the contagious period of syphilis varied in different patients. The time during which the typical signs of contagion appeared and recurred might vary from three to four years. During this time syphilitic patients ought not to be allowed to marry. A certain number of facts, luckily rare, proved that syphilis might be transmitted after ten years. The lesions by which these late transmissions were produced were generally eruptions of simple appearance. Among the causes which ought to be mentioned as important is tobacco. Most patients by whom contagion was transmitted were smokers and had sores in the mouth. Early treatment did not seem to have any influence on the virulence of prolonged syphilis.

Malignant Syphilis.

Professor HASLUND (Copenhagen) considered that malignant syphilis was a purely secondary form of syphilis, and had nothing to do with tertiarism. It had been confused with other forms, especially with tertiary ones, from which it ought to be clearly distinguished. The name was objectionable, partly because it had been applied to various forms differing much from each other, and partly because it could not in reality be called "malignant" in the sense in which this term was used for other diseases. The prognosis was comparatively favourable. Among 8691 cases of syphilis treated in the Copenhagen Municipal Hospital during fourteen years, malignant syphilis had been observed thirty-nine times, and with equal frequency in men and

women. It was impossible to lay down definite rules for the treatment ; this must vary very much, and must be governed by the general condition of the patient, the gravity of the symptoms, the previous treatment, and the general history of the case.

Professor NEISSER (Breslau) considered that "syphilis maligna" differed from "syphilis gravis" and "syphilis anormale grave" in the characteristics peculiar to the disease. The characteristics of syphilis maligna were the general and severe symptoms, and the numerous, early-appearing, frequently relapsing, large pustular and ulcerating forms. The haemorrhagic forms of syphilis were only to be considered as complications, and in this sense scurvy formed a severe complication in syphilis. The multiplicity and wide-spread occurrence of the eruption was a further characteristic of syphilis maligna, which was not a tertiary-gummatous form. The reasons for this were that the ulcerations were not serpiginous, and the action of iodine salts on the disease was uncertain. He was inclined to think that the malignant form resulted from some special susceptibility of the diseased individual, and there were no grounds for thinking that syphilis maligna was due to a specially virulent poison. The presence of Staphylococci in the ulcerated tissues was a secondary occurrence. The anatomical position, nature and course of the primary sore had nothing to do with the occurrence of syphilis maligna, and specific treatment was often unsuccessful. Tonic treatment and external sulphur treatment were, however, often useful. Syphilis maligna could be acquired or inherited ; the prognosis was good. In the ætiology of syphilis maligna, mercury treatment played no part.

Prognosis of Extra-genital Syphilis.

Dr. ERNST FEIBES (Aix-la-Chapelle) said that it was generally held that such infection carried a worse prognosis than genital infection, but was that the case? The accompanying buboes were often found extremely developed, and in consequence of non-recognition of the primary sore, and perhaps of consequent delay of treatment, the secondary symptoms were frequently severe. Relapse was not more frequent than in genital infection, and probably no greater proportion of tertiary syphilis occurred than in the case of genital

infection. He had observed forty-five cases, and they showed that extra-genital infection did not cause more serious consequences than genital.

Tuberculosis.

Dr. H. HALLOPEAU (Paris) thought that tuberculosis could produce skin diseases in a direct manner by the action of its contagion or of its toxins. It was important to be able to recognise the tuberculous character of any case as early as possible. The following points were decisive : The possibility of transmitting tuberculosis by a series of inoculations of the morbid matter, the appearance of tubercular bacilli in the infected tissues, the production of alterations by intradermoculture and the production of different eruptions by the action of tuberculin. The clinical types, which (besides lupus vulgaris) had been up to the present time associated with tuberculosis were—(1) Sclerotic and verrucous tuberculosis ; (2) primary or secondary ulcerative tuberculosis ; (3) intra-dermic growths of osseous, lymphatic and subcutaneous origin ; (4) erythematous lupus, and (5) anatomical tubercle.

The first type owed its characteristics to having its seat in the papillary body. In the second type—
a. Primary and secondary ulcerating tuberculosis originated round ulcers in patients suffering from phthisis.
b. Extensive ulcerations, which often complicated other forms of tuberculosis, such as osteitis, gummatous growths, and lupus vulgaris. These ulcerations might bear some resemblance to verrucous ulcers or phagedænic conditions. In the third type, in most cases the skin was only passively affected by these subcutaneous growths ; but this was not always the case ; the fistulous tract might, according to the various degrees of susceptibility to infection or vulnerability of the different elements of the skin, become the starting point, either of lupus vulgaris or verrucosis, or of progressive gummatous growths. In some of these forms the new growths became modified, and might lose their tendency to undergo caseation. In the fourth type, this lupus might rise to adenopathy in the surrounding parts, the tuberculous nature of which could be demonstrated. It might be associated with lupus vulgaris, although bacilli have been but rarely found. Tuberculin injections produced reaction, and the existence of anatomical changes similar to

those of tuberculosis had been proved. In the fifth type (*a*) the tuberculosis produced by inoculation appeared under different forms according to the liability of the tissue to become a favourable medium for the growth of the virus; (*b*) these alterations offered, at one time or another, pathological forms of different histological nature.

Dr. H. RADCLIFFE CROCKER (London) urged that, while there were a certain number of diseases directly due to the presence of the tubercle bacillus, there were many other diseases of the skin which, while not directly due to this organism, found a favourable soil in those persons who are easily invaded by the bacillus, especially in those who presented the phenomena of easily excited suppurative bone disease, lymphatic gland enlargement, &c., comprised under the clinical term of Scrofula.

Keratosis.

Dr. UNNA (Hamburg) thought that in the normal keratoid cells the keratosis was confined to the external cell membrane; the nuclei, the keratohyalin and the eleidin were in no way implicated in the process of keratosis. Chemically, keratin was very closely allied to protoplasm, being chiefly distinguished from it only by its containing a higher percentage of sulphur, and by yielding more tyrosin in the process of decomposition or differentiation. The keratosis of protoplasm was probably set up through the influence of materials brought to the epithelial cells by way of the lymphatic vessels. In like manner, indigestible protoplasm could be produced from digestible protoplasm. Pathologically considered, there were to be specially distinguished three types of keratosis, viz. Callus, Psoriasis, and Ichthyosis. In these hyper-keratoses, the proportion of keratin did not correspond to the degree of induration clinically observed. Pathological keratosis was a marginal or superficial keratosis. The piling up of horny masses, which characterised all hyper-keratoses, did not depend on an increased proportion of keratin in the individual cells, but on the aggregate amount of keratin they contained collectively, and showed itself in a smoothing down of the surface reliefs of the horny cells. The study of the relief of these horny cells was of great importance in the pathogenesis of hyper-keratoses.

Prurigo.

Dr. ERNEST BESNIER (Paris) considered that the pruriginous affections which Willan brought together into the class of papular affections—viz. Strophulus, Lichen, and Prurigo—constituted a natural and normal dermatological group for which the denomination of the *Prurigo Group* was perfectly appropriate. The name Prurigo, with suitable epithets, was capable of representing all the affections included under this single heading in the most exact manner and without the slightest ambiguity. The adoption of this terminology, while re-establishing the unity of the descriptive substantive word (Prurigo) would at the same time allow them to put an end to the existing confusion which resulted from the multiplicity of species, of forms, and of varieties, now arbitrarily named according to characters purely morphological. The word Pruritus, according to its exact significance, corresponded to a symptom common to a large number of morbid states, and could not appropriately represent a syndrome or group of symptoms, still less a disease. Its meaning as the mere description of a symptom ought to be restored and restricted as such. The group of Prurigos, thus re-constituted, included the genus Prurigo of Willan, and those embraced under "Strophulus" and "Lichen" which, since Hebra's time, had been unjustifiably included under various forms of the Erythemas, Urticarias, and Eczemas. And, as a matter of fact, the majority of these forms, revised and studied anew in accordance with the necessities of modern dermatology, had already been restored to the group of Prurigos; they had only to complete this order of things. The attempt, still made by some eminent dermatologists, to erect a new and definite type of Prurigo, based upon an anatomical character supposed to be specific, would (were it successful) only permit the confusion to persist which he had indicated with regard to affections of the same pathological order as the Prurigos, and which, therefore, would still be classified under the separate headings of the Erythemas, Urticarias, and Eczemas. Many sorts of true Prurigo—*pruriginous, multiform, chronic, relapsing, dermatites*—had multiple and commonplace (*banal*) lesions as their anatomical basis: in the front rank of these were "*lichenisation*" and "*eczematisation*," which formed an important class among those Prurigos which

were dependent upon diathetic conditions inherent in their subjects ; they constituted one of the commonest types of *diathetic prurigo*. General neurology and dermatological neuropathology were not sufficiently advanced to permit of the thorough discussion of the question of angioneuroses, neurodermias, and neurodermites as applied to the theory of the Prurigos. Whatever the pathological factor might be which dominated the production of pruritus and of the primary or secondary skin manifestations of the Prurigos, the previous existence of direct or indirect *blood changes*, of a very complex character, seemed to be extremely probable. According to this view, Prurigos would be considered as *toxidermias* or *autotoxidermias*, temporary, intermittent, remittent or permanent. Whether the specific element was toxic or toxicin, whether it existed in the blood or in the lymphatic spaces by reactions either secondary, provoked or spontaneous, the irritant acted upon the sensory centres in the spinal cord or on the peripheral nerve-endings, and produced along with pruritus—or as its result—troubles of circulation and of nutrition which represented the series of primitive tissue changes. Pruritus in prurigo preceded and dominated the lesions. The pruritus connected with the papule or with the other anatomico-pathological changes present must not be confounded with the local, pre-existing pruritus, nor with the diffused pruritus which extended a considerable distance outside the area and limits of the actual lesion, and which was the direct result of the primary irritation of the cord-centres or of the peripheral nerves, and not of the lesion itself, which was, as it were, accidental or contingent. The pruritus survived the papules ; the papules never survived the pruritus.

In prurigo the direct action of the pathogenic conditions was confined to the production in the skin of a physio-pathological condition, of a disturbance of sensation and nutrition, and, in the acute forms, of a more or less marked degree of neuro-vascular tension ; but it did not generally bring about surface lesions by itself, nor at the onset. These surface lesions, whether acute, subacute, or chronic, primary or secondary, immediate or dissociated, included (among their essential, almost always necessary factors) the traumatic acts of scratching, &c.

The skin, if absolutely and appropriately pro-

tected from "trauma" of every sort, remained entirely free from surface lesions.

Dr. JAMES C. WHITE (Boston, U.S.A.) considered Hebra was the first to establish the individuality of prurigo, a disease which had its true home in Vienna.

Prurigo must be placed amongst skin diseases of well-established character and recognised in Europe, but true prurigo was still an extremely rare disease in America. Its frequency was greatest in the country which, of all Europe, presented the greatest diversity of races. Defective nutrition, bad hygienic surroundings, and negligence as regards the skin were the only positive factors which had been recognised as bearing on the etiology of the disease. The pathology of prurigo was unknown, and opinions differed widely as to the primary manifestations, the anatomical character of their so-called characteristic lesions, and their pathological significance. Personally, the author was of opinion that there existed in early childhood a condition allied to pruritus and urticaria, which in certain parts of the world often became chronic, owing to inexplicable national cutaneous traits or inherent custom of living.

Dr. J. F. PAYNE (London) thought that prurigo was distinct from pruritus or itching. Pruritus was a quite peculiar functional disturbance of the cutaneous nerves, not a mere exaggeration of common sensibility or of the sense of pain. It differed, therefore, from hyperesthesia, hyperalgesia, and reflex irritability. The impulse to scratch was not purely reflex, but obscurely volitional ; there was an irritable condition of cerebral centres as well as of the cutaneous nerves. In true prurigo there was an anatomical change in the skin, as well as deranged sensation. The essential anatomical element was the "pale papule" which was found in all cases of true prurigo, and also occurred in some cases which began as functional pruritus. Prurigo was essentially a chronic morbid condition of the sensory nervous system, central and peripheral, which might be induced by pruritus and scratching, but conceivably be sometimes primary. The papular eruption was not the cause but the consequence of this condition. There was no positive evidence how the nervous disturbance produced papules, but probably it was merely by scratching. In

severe prurigo they must assume some special susceptibility of the nervous system. The term prurigo should not be confined to the severe form described by Hebra, which was extremely rare in England, but included prurigo mitis vel simplex. The affection called infantile prurigo or lichen urticatus was closely allied to this, being not solely due to urticaria.

NOTES AT THE OXYGEN HOSPITAL, 35, ST. GEORGE'S SQUARE.

The Oxygen Treatment.

THE essence of this treatment consists in the exposure of the affected part to the continuous or intermittent action of oxygen gas, either pure or diluted, the diluent used being purified air.

In the female ward, case No. 1 was a girl, D. M., æt. 17, who came in on the 11th of May; she had previously had the oxygen treatment for 4 months, for a large ulcer in the centre of her back. The ulcer originated from a burn received in November, 1894. She was treated in one of the London Hospitals for eight months. Before the first application of the oxygen treatment the ulcer measured 10 in. by 11 in., and after four months of this oxygen treatment the ulcer was reduced to 3 in. by 1½ in. In October, 1895, the treatment had to be abandoned, and was not resumed until the 11th of May. In the interval the ulcer had somewhat enlarged, and on admission into this hospital, three weeks ago, the ulcer measured 4 in. by 3 in. After three weeks' treatment the ulcer was now improving, and the granulations were looking much healthier, and the ulcer was evidently healing. Some six weeks ago she received, in addition, two severe burns, one on the right hand and the other on the right thigh. On the right hand the ulcer extended from the centre web, between the first and the middle fingers, to the under side of the little finger, measuring on her admission here, 4 in. by 4½ in. The wound was foul and unhealthy looking, and emitted a most offensive smell. The hand was placed in the oxygen apparatus and treated with a quarter mixture of oxygen and air. In two or three days the smell

entirely disappeared, and the surface of the wound became healthy and covered with granulations, and after three weeks the healing had extended as far as the ulnar side of the middle finger, and was about half the size it was on admission—the exact measurements being now 1¼ in. by 1½ in. The burn on the thigh was oval in shape, and on admission was covered with large, unhealthy, flabby granulations, which bled on the slightest touch or on exposure to the air. The wound was enclosed in an apparatus composed of a glass cylinder with india-rubber funnels fitting above and below, and the wound was treated with a quarter mixture of oxygen and air. This had now been a fortnight under treatment, and there was a margin of new skin all round the wound, in some places half an inch wide, and it was rapidly extending. The granulations were much healthier, and they did not bleed. In both these recent wounds, almost instant relief from pain was afforded by the application of oxygen; this is one of the most marked features in this treatment, pain being relieved at once. It was further to be remarked in reference to this case that none of the cicatrices showed the least sign of contraction, and the cicatrices in the hand were mostly of normal skin.

In the male ward were shown two cases—one a case of suppurative middle-ear disease occurring in a young man of 22; his history was as follows:—Four years ago he was attacked with acute inflammation of the middle ear, which ended in suppuration and perforation of the membrana tympani.

The discharge continued profusely in spite of all treatment, and in last January the mastoid antrum was trephined in the military hospital at Bombay. Although the discharge slightly diminished after the operation, it never entirely ceased. Three weeks ago he was admitted here, and oxygen applied five hours daily for periods of half an hour at a time, the mixture used being oxygen and air in equal parts, the end of the tube attached to the gas bag being placed into the external auditory meatus. His hearing distance for the ticking of a watch on entrance was $\frac{1}{2}$ of an inch; he could now hear 2½ in. distant, and the discharge had entirely ceased.

The second case was a boy æt. 17, a Militia recruit. Many years ago he received a severe scald on the front of his right leg; about twelve weeks ago he received a kick on the shin on the old cicatrix left by the scald. He was treated in

the military hospital for two months with many forms of treatment, but without any favourable result. On admission here the wound measured $2\frac{1}{4}$ in. by $2\frac{1}{2}$ in. He had been two weeks under treatment, and the wound measured $1\frac{1}{2}$ in. by $1\frac{1}{2}$ in. It was very painful on admission, but this pain at once disappeared on the affected limb being placed in the oxygen apparatus.

The oxygen used in the Hospital is from Brin's Co., and is kept in steel drawn cylinders, which appears to be the safest way of keeping it. The oxygen is never used pure except in cases where the skin is unbroken, as it is too irritating. The usual strength used is a quarter of a foot of pure oxygen to three quarters of purified air, the bag used in the treatment containing a cubic foot. The best plan is to fill the bag with the required amount of oxygen, and then fill up with purified air passed through two wash-bottles, the first containing lime water, and the second a solution of permanganate of potash.

With regard to the dressing of these wounds, nothing is used but boiled water, with which they are washed twice daily.

It is necessary to observe, however, that the oxygen causes the formation of a parchment-like film around the edges of the wound or ulcer, and it is necessary to carefully remove this once daily, as, if allowed to remain closely attached to the healing edge, it prevents the access of oxygen and delays the process of cure.

THERAPEUTICAL NOTES.

A Method of Curing Tic Douloureux.—

Dr. Charles L. Dana, at a meeting of the Association of American Physicians, of New York, referred to the rebelliousness of these cases to medicinal and even to surgical treatment. One patient, who had had the Gasserian ganglion removed by a surgeon, suffered not only a recurrence of the pains, but became insane. The method by which he and Dr. Elliot, of New York, had cured seven out of eight cases consisted in hypodermic injections of very large doses of sulphate of strychnine. Rest abed was enjoined during the treatment, and freedom from care. Strychnine injections were given once a day, beginning with, say, $\frac{1}{60}$ grain (0.002 gramme) and rapidly increased to

$\frac{1}{4}$ or $\frac{1}{2}$ grain (0.012 or 0.015 gramme). Patients could not usually take more than $\frac{1}{4}$ grain (0.015 gramme). The large doses had a peculiarly anodyne effect, quieting the patient for hours like morphine. Usually the injections were discontinued after five or six weeks, but might have to be renewed once or twice again. On discontinuing it he gave iodide of potassium, nitro-glycerine, or other drugs. In a former paper he had impressed the importance of arterial sclerosis in neuralgics. As this treatment had only been begun two years ago, he was hardly justified in speaking of the result as a cure, yet it had been in such contrast with that obtained by other methods that he wished to emphasise its value.—*Universal Medical Journal.*

The Tonsillar Cough.—According to Dr. Furet, this cough may result from any pathological alteration of the tonsils. It was sufficiently explained by the complex innervation of the gland. In fact, the glosso-pharyngeal, the lingual, the spinal, and the pneumogastric nerves were blended and became entangled at their outer surface, where they formed a small plexus, which Andersch had described under the name of the tonsillar plexus. It must not be forgotten that the tonsils were enclosed by the muscles of the pillars of the fauces, which were very distinctly connected with the muscular apparatus of the larynx. Tonsillar cough was violent, spasmodic, and even extremely painful. It was frequently accompanied by reflexes in the neighbouring region, and particularly by watering of the eyes. It was distinguished from the cough due to affections of the respiratory tract by the complete absence of expectoration, and, owing to this fact, it did not yield to any of the remedies generally used.—*Record.*

The Treatment of Sepsis by Intravenous Serum Injections.—Berlin reported to the Société de Chirurgie the case of a woman who, three days after vaginal hysterectomy, showed such violent symptoms of sepsis that recovery seemed absolutely impossible. The belly was distended and painful, and there was very marked subnormal temperature and frequent vomiting. On the fourth day the patient seemed to be about dying, whereupon Berlin gave an intravenous injection of a little over a pint of artificial blood-serum, and shortly afterward repeated this injection, using not quite two-

pints. Marked cyanosis and increased gravity of symptoms followed at once, but shortly there was decided improvement and the patient recovered, being completely convalescent the twelfth day of her illness.

Segond stated that he had used injections of artificial serum for four years in all major operations, and had been able to save many cases of severe haemorrhage by this means. He also stated that in cases of severe sepsis these injections were potent.

Monod had also had excellent results from intravenous injections.

Michaux in severe haemorrhage and in cases of peritoneal sepsis had given intravenous injections of about three pints of serum, usually with excellent results. Sometimes immediately after the treatment serous fluid was vomited in large quantities.

Terrier stated that it made no difference whether the serum was injected intravenously or subcutaneously, and held it was wise to combine such injections with blood-letting.

Therapeutische Wochenschrift.

Craniotomy for Idiocy.—Dana, in an able paper on this subject, lays down the following indications for operation :

Very little hope can be entertained after a child has reached the age of four or five years, and the best chances for improvement will be obtained if the operation is done under the age of four. It has been shown that the development of the brain is divided into three equal periods: The brain increases in weight by one-third during the first three months of life; an additional increase of one-third takes place between the tenth month and about the middle of the third year; and the final increment takes place during the period between two and a half years and adult life, chiefly before the age of seven years. It is in the second third of the brain development or the early part of the last third that most improvement is recorded from craniotomy. At the same time there have been some striking successes, so far as symptoms are concerned, in children as old as nine or eleven years, but these were cases in which there had been epilepsy or paralysis, or some other motor disturbance. The simple forms of idiocy with microcephalus are certainly not benefited unless operated upon early in life.

The operation so far shows that it is in the simplest forms of idiocy, with a moderate amount of microcephalus, that most improvement is to be expected. Children who have birth-palsy and decided double paraplegia, and children who have a hemiplegia with epilepsy dependent on some extravasation of blood and large sclerotic focus in the brain, are rarely helped. It has, however, occurred that even in this latter class of cases improvement has been brought about so far as the epileptic attacks were concerned.

Before operating or selecting a patient for operation the question of cretinism should be carefully considered. If the child is a cretin—that is to say, if the idiocy is due to myxedema following upon the absence of the thyroid gland—then no operation should be for a moment considered, but the child should be fed upon thyroid extract.

The question of hereditary syphilis should be carefully considered, particularly in the first months or year of life, and if there are decided indications of syphilitic exudation or of destructive change in the brain, vigorous mercurial treatment should be attempted before anything else is undertaken. The idiocy due to a hereditary syphilis, meningitis or meningo-encephalitis is not likely to be helped by operation if medical therapeutics fail.

A good many idiotic children have manifest signs of rickets or tuberculosis. In the case of rickets the operation is not necessarily contraindicated, but the child should receive proper nourishment and medical care before it is attempted. In distinctly tubercular children it would be folly to attempt any surgical interference.

There seems to be a very great difference among observers as to what constitutes microcephalus. There is generally a rather small head in these cases of idiocy, but distinct microcephalus is rare. When it exists it is doubtful if operation does any good, because here there is usually very great lack of brain development.

Dana appends a table in which he shows that the operation of craniotomy for idiocy is still justifiable in a certain selected class of cases. He shows that by perfected methods of operating the danger to life is under five per cent., and will probably become still less. The clinical reports quoted show improvement after operation too often for the facts to be ignored, even allowing for the happy therapeutic temperament of the operating

surgeon ; and in his personal experience one-half of the recovered cases have been improved.

The method by which improvement is brought about is largely a surgico-pedagogic one. All those who have had experience in educating idiots lay stress on the importance of special pedagogics ; and craniotomy belongs, in a measure, to this class of therapeutic procedure. It may have some additional value by stimulating the circulation and nutritional activity of the brain.

The cases in which operation is indicated are generally those of cerebral agenesis, rather than those having extensive sclerotic lesions and palsies. The operation may sometimes be repeated with benefit ; and the results are not always immediate, especially so far as the cessation of the fits is concerned.

American Journal of the Medical Sciences.

Comparative Value of Remedies for Uric Acid.—F. LEVISON (*Ugeskrift for Læger*) has examined the new remedies recommended as solvents for uric acid, not only for concretions in the kidneys, but for the form of uratic deposits in gout. Evidence from literature, as well as his own experience, leads him to the conclusion that neither piperazine, lycetol, nor lysidin had any special remedial property in this affection. They combine readily enough with uric acid to form soluble salts, but when given *per os* and excreted with the urine, they lose that property completely or in greater part. Another new remedy, urocedin—recommended by Mendelsohn—is a combination of citrate of sodium and sulphate of sodium, with small quantities of citrate of lithium and chloride of sodium, and is not superior to the citrate of sodium so long in use.

More remarkable results are obtained from urotropin, introduced by Nicolati. This remedy can be taken in a dose of 1 to 2 grammes ($15\frac{1}{2}$ to 31 grains) a day. It is rapidly excreted by the kidneys, and can be demonstrated by bromine water, which gives a yellow crystalline sediment.

Levison found that the urine of a person who had taken $1\cdot5$ grammes (24 grains) of urotropin was able to dissolve small quantities of uric acid by passing through a filter covered with $0\cdot50$ grammes ($7\frac{3}{4}$ grains) of uric acid ; when the uric acid was digested for twenty-four hours with the urine containing urotropin, as much as 16 to 18 per

cent. of it was dissolved. Concretions of uric acid treated with this urine were somewhat diminished in weight, and their surface became uneven and greyish instead of yellow. Still another property is conveyed to the urine by urotropin,—it is rendered bactericidal and aseptic. The urine could be held for many days in the thermostat at a temperature of $37^{\circ}\text{C}.$ ($98\cdot6^{\circ}\text{F}.$) without becoming foul, even when different cultures of bacteria were added to it, while samples of urine without urotropin fermented and putrefied in a very short time. Urotropin seems thus to be indicated in suppurative diseases of the kidneys and bladder ; it must, however, be observed that in one experiment the urine containing urotropin began after some days to precipitate a crystalline sediment of phosphate of lime ; if such a precipitate took place in the living body it would, of course, be a serious inconvenience in the use of the remedy.

The Removal of Cerumen from the External Auditory Canal.—In a practical note upon this subject we are told by Laurent that under no circumstances should cerumen be removed from the ear by the use of metal instruments, such as tweezers, stylets, or similar instruments, which may do damage, unless the physician be an experienced otologist and familiar with the technique of removing foreign bodies from the ear ; for by the use of these instruments, aside from the ordinary damages which may be done by bruising, it is possible to produce furunculosis of the external auditory canal through infection of the glands, and there is also danger of injuring the tympanic membrane, or of causing haemorrhage from the canal, or deafness and vertigo. By far the safest and most efficient plan for the removal of wax under these circumstances is the use, by means of an absolutely sterile syringe, of water which has been sterilized by boiling and which is as hot as the patient can bear. If desired, a very small portion of carbolic acid may be added for its antiseptic influence. After the water is sucked up into the syringe, care should be taken that the point of the instrument is elevated and that all air is driven out of it, as the injection of bubbles of air into the external auditory canal produces a very disagreeable and painful sensation in the ear. A small pitcher or vessel, usually used for receiving liquid when the ear is being irrigated,

is held tightly against the skin beneath the ear, and then the water is injected with gradually increasing force until the mass is loosened and readily removed. Great care must be exercised that the first drops in particular are not driven into the ear with too much force, for if force is used vertigo and pain will result. In other words, no forcible measures should be used under any circumstances for the removal of such accumulations as we are speaking of. Five or six syringefuls of water are usually sufficient. In other instances irrigation by means of a fountain syringe held eighteen inches or two feet above the ear of the patient is more efficient than the piston syringe. If the mass in the ear is so hardened that the injection of water will not soften it, it is well to prescribe as follows :

Bicarbonate of sodium, 15 grains ;
Glycerin, 1 drachm ;
Water, 1 drachm.

Three times a day the patient drops into the ear by means of a spoon, or with a medicine-dropper, five or ten drops of this solution, which has previously been warmed, and after it has remained there for a few minutes a pledget of cotton should be placed in the external auditory canal in order to preserve the moisture. In the course of a day or two the cerumen will be so softened by this solution that it will be easily removed. If it is perfectly free in the auditory canal it can, of course, be removed by a scoop or tweezers, but these are never to be employed unless the concretion is already loosened. After the removal of the mass the ear should be thoroughly dried by means of absorbent cotton, and a small pledget of absorbent cotton be placed in the external auditory canal to protect the part from cold. If these measures are used, serious results never occur, and the deafness which has been produced by the obstruction in the canal speedily disappears.—*Journal des Practiciens.*

REVIEWS.

The Phonographic Record of Clinical Teaching and Medical Science. London : Sir I. Pitman & Sons, 1, Amen Corner, E.C. Price 4d.

Dr. Scott Riddell, of Aberdeen, and Dr. Gowers

are responsible for the greater part of the June number, and in the six cases of remarkable injuries related by Dr. Riddell, is one where a fall upon the end of a crowbar caused a wound of the scrotum, which after being cleansed and sutured, healed kindly. The patient did well till the fifth day, when, after a purgative, rapidly fatal peritonitis came on. At the necropsy a portion of the man's drawers and trousers were found in the abdomen, over the stomach, the crowbar having traversed the inguinal canal, carrying the clothing with it into the abdomen. In another instructive problem in diagnosis, in a case of pontine lesion, Dr. Gowers says the first thought in a case of hemiplegia should be, what is the lesion ? and in determining this the state of the heart and vessels is generally a sufficient guide ; their condition may be such as will burst an artery, or, on the other hand, favour the formation of a clot or a deposit of an embolus in a branch. Prodromata which indicate an abnormal state before the seizure point to softening.

Deep coma during the first twelve hours suggests haemorrhage. Turning to the question of the seat of the lesion, Dr. Gowers points out that disease in the pons has to extend very little to cause great danger to life ; that a lesion below the middle of the pons causes "alternate hemiplegia ;" and one above the middle of the pons and the decussation of the motor path for the face, facial paralysis on the same side as that of the limbs ; whilst, in addition the movements of the eyes are frequently interfered with, the ocular centres being beneath the corpora quadrigemina.

Disease of the pons has also a peculiar tendency to disturb respiration and produce hiccough, and cough.

Dr. Gowers also contributes a practical paper on Fallacies in Auscultation. Friction in the shoulder joint may be mistaken for dry pleuritic rub ; a diastolic exocardial murmur may sometimes be heard, especially when there is a depressed sternum, and is to be distinguished by the effect of posture ; a respiratory murmur of cardiac rhythm may puzzle, but ceases if the patient holds his breath ; whilst a subclavian murmur, always heard on both sides, is of no importance so long as we know that it may occur. Dr. Gowers makes the following rules : When there is an abnormal heart sound in the erect posture, make the patient lie down and examine again, and always identify the first cardiac sound by the carotid and not by the radial pulse.

For gall-stones Dr. Gowers suggests the use of succinate of iron in doses of ten grains twice daily for some months.

In six cases Dr. Gowers has found its use followed by disappearance of all symptoms, the good effects lasting in one or two cases for ten or twelve years.

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NOTES FROM THE CLINIC

OF

MR. PEARCE GOULD

In the Wards of Middlesex Hospital.

Stricture.

OUR first case to-day is a man who has been admitted for an impermeable stricture of the urethra. This patient is aged 42 : he suffered from gonorrhœa four years ago, and since then he has had a gleety discharge which has continued up to the present. In 1892 he went into an infirmary because of pain in passing urine, and was treated for a stricture. After this the patient himself passed bougies until sixteen months ago, but stopped then because of the pain, and has not had any treatment since. Six months ago he sustained an injury to his right testicle. Sometimes his urine has dribbled away involuntarily. On admission to the hospital, the smallest bougie could not be passed ; his urine dribbled from him, and was alkaline and opalescent, but contained neither blood, sugar, nor pus. Such is the patient's history. Before examining the urethra we will carefully examine the testicles. On the right side the epididymis is found in front of the testicle, and it is enlarged and hard at its lower end. You notice that the testicle hangs very loosely in the scrotum, so that it is easy to turn it round, and you can also turn it upside down. This very free mobility of the testicle may leave you in doubt as to whether the organ is really lying with the epididymis in front, or whether the testicle has been turned round by the examining hand. There is a way of correcting any error arising from this mobility ; it is to carefully feel the cord and notice the relative position of the vas deferens and the vessels. When the testicle is inverted the vas is found in front of the vessels, but mere twisting round of the testicle does not affect the cord. In this man if you take the right cord in your left hand, and the

left cord in your right hand, you will find that as you pass the fingers from behind forwards, the last structure the left hand meets anteriorly is the vas in front of the right cord. On the left side, the vas is felt at the back of the vessels. In the left testicle also you notice there is induration in the globus minor, but the organ is normally placed in the scrotum. He has, therefore, an inverted right testicle with a considerable swelling of the globus major, a normally placed left organ, and evidence of chronic induration of the epididymis on each side.

Now for the principal point in this case. The man has been admitted for a stricture of the urethra, through which even the finest bougie could not be passed in the out-patient room. There is no complete retention of urine, but, on the contrary, urine is more or less continually dribbling from him. "Incontinence of urine" is an ambiguous term ; it is generally used to denote either great frequency of micturition, or inability to control the flow of urine ; incontinence literally means a loss of power to retain urine. In tight strictures dribbling of urine is very frequent, and it is interesting to notice both the fact and the explanation of it. Urine is retained in the bladder partly by muscular action and largely by atmospheric pressure ; eight inches of the urethra is kept closed, and the sides, moistened with mucus, are kept in apposition ; this is a very effective valve. Urine is held in then by the contraction of the circular muscular fibres at the internal orifice of the urethra, by the contraction of the compressor urethrae muscle, and largely by the atmospheric pressure upon the walls of the urethra. When there is a tight stricture the thickening and hardening of the urethral wall forms such an obstruction to the escape of the urine that the constant action of the sphincters becomes less and less necessary, until at last they cease to act altogether ; as it were, they refuse to do unnecessary work. We see the same thing in the patulous anus below a tight stricture of the rectum. In long-standing strictures of the urethra, the muscular fibres of the bladder hypertrophy,

and there is a stronger expelling force, and when retention of urine is superadded, and the bladder is chronically full, this powerful bladder overcomes very easily the atmospheric pressure, and squeezes a drop or two through the stricture. The dribbling of urine, wrongly called "incontinence," is therefore seen in cases of tight strictures of some standing, and where the bladder is not regularly emptied.

The next point I want you to notice is of great practical importance; it is the value of rest in the treatment of stricture.

This man came in ten days ago; at that time two unsuccessful attempts were made to pass fine bougies through the stricture. There was nothing urgent in his condition, as the urine was dribbling away and relieving the retention. So for ten days I have done nothing but to keep him in bed and attend to his bowels, but we hear from the notes that the passage of the urine is much easier and freer now, not mere dribbling, but a voluntary passage of a fair stream. This is a common result of mere rest, and I want you to remember that whenever you are called to a case of tight stricture, if there is not complete retention calling for instant relief, simply exercise patience and put the man to bed, keep him warm, on simple unstimulating diet, get his bowels freely open, and by doing that the condition of the stricture will certainly improve, and in a few days you will be able to pass an instrument. Supposing I am able to pass an instrument through this stricture now, the inference is not to be drawn that we are more lucky or more skilful than those who made an unsuccessful attempt ten days ago, but that by keeping the man in bed ten days or so the condition of his urethra has improved. The rest leads to subsidence of spasm and of congestion, possibly also to absorption of some of the stricture tissue; but whatever the full explanation may be, there is not the smallest doubt whatever about the fact. In tight, difficult strictures, unless there is retention, do not worry and irritate the urethra by frequent attempts to pass instruments, which excite spasm, and cause additional congestive swelling of the parts, but give the man rest, and leave the urethra alone for some days, and then you will almost certainly be able to pass an instrument. So true is this that some years ago there was a surgeon in one of our London hospitals who regularly treated stricture

by rest and nothing else; he did not use instruments at all. He did it chiefly to point out the value of rest.

This influence of rest explains why you obtain better results in the dilatation of stricture in patients taken into the hospital than in those treated as out-patients.

We have had this patient's urine measured, and the daily amount passed has been from 50 to 60 oz., with a specific gravity of 1022. When he came in it was alkaline and turbid, but there was no blood. By holding a piece of moistened red litmus paper above the urine, we found it turn blue—the urine was ammoniacal. The interpretation of the chemical fact that the man had ammoniacal urine is that the urea has been broken up into water and carbonate of ammonia, as a result of fermentation, and the cause of that fermentation is the presence of micro-organisms, such as the *Micrococcus urea*. We naturally ask the question whether in any patient who does not periodically empty his bladder the urine would become ammoniacal. With chronic retention, such as this man has had for weeks, there is always some residue left in the bladder, but this alone is not enough to lead to urinary decomposition. You will often see men with large prostates and bladders up to the umbilicus, the result of retention and incomplete emptying of the bladder, which has lasted for months, and sometimes for years, whose urine is quite clear and acid. There is something more than retention required; it is an important factor in the case, but simple retention of the urine does not lead to decomposition of the urine. Another element in the causation is the state of the mucous membrane. The most striking example of this is seen in cases of fracture of the spine and crush of the cord with loss of nervous control over the blood-vessels of the bladder, and consequent nutritive changes in the mucous membrane. Cystitis and decomposition of urine generally quickly follow upon such an injury. And so if a man's general health becomes undermined—and that occurs often in the subjects of old tight stricture—there may be some obscure nervous change which allows this fermentation process to take place. Perfectly healthy mucous membrane has an inhibitive power over the fermentation process. But given an unhealthy mucous membrane, produced either by inflammation or by

obscure trophic change, then the micrococcus is able to grow and cause this breaking up of the urea into water and carbonate of ammonia. We must not forget, too, that the frequent powerful contraction of the muscular coat of the bladder trying to force the urine through the narrow opening at the stricture, causes congestion of the mucous membrane, and frequently recurring congestion of the mucous surface is an important predisposing cause of cystitis. It has also been pointed out that in tight strictures with dribbling, urine lies in the urethra, which ought to be quite closed. This urine thus lying in the urethra decomposes very rapidly, and it is said that this decomposition spreads back through the stricture to the urine behind. However this may be, the fact remains that if a man has a tight stricture with considerable chronic retention, it is commonly, though not invariably, the case that cystitis occurs. We have treated this man with boric acid 10 gr. and 10 minims of tincture of nux vomica in an ounce of camphor water, and his urine is now much better than when he came in ; it contains less pus, and is only just alkaline in reaction. The importance of the specific gravity of the urine is that it affords an index to the state of the kidneys, indicating whether they are purifying the blood of the urea and other solid matters. When a man has a tight stricture of the urethra, chronic inflammatory changes, which gravely impair their excretory function, are very liable to be set up in the kidneys. And when to tight stricture is super-added cystitis, the liability to chronic changes is greatly increased, and there is danger also of acute nephritis in addition. It is important not only in regard to treatment, but also in regard to prognosis, to know whether we are dealing with a man with sound or unsound kidneys. This patient's amount of urine is a little more than usual, but the specific gravity is 1022, and there is no *prima facie* reason whatever to doubt that he is excreting urea fully, and that his kidneys are fairly natural organs. The precise estimation of the amount of urea is the best way of determining this point. Before passing an instrument and examining the urethra, we will ask about this accident to the testicle. He says that six months ago he was nipped in the testicle while at work on a drain ; he felt sick, but no swelling was noticed at this time. We may, of course, read his statement in one of two ways ; he

may have had a normal organ, which was nipped as he was stepping down into the drain, or he may have had a large and tender organ, which, from a slight squeeze, became very painful and enlarged. That seems the more probable view, as just stooping down into a drain or trench would not cause a squeeze likely to seriously damage the testicle. It seems more likely that the testicle was damaged before the slight squeeze determined the pain. And when we find that it was the epididymis and not the body of the testicle that suffered, the probability becomes a certainty, for in injury of the testicle it is the body of the organ that suffers. The connecting link between the tight stricture and the epididymitis is the gleet of four years' standing ; inflammation spread from the urethra down the vas deferens to the epididymis. Otis spoke of gleet as being the signal of stricture, he said that it was a flag held out to indicate stenotic trouble. It is a fact that with a tight stricture you have a gleety discharge. This has been explained by the irritation caused by the urine being driven by a powerful bladder through a narrow orifice. The friction at the stricture causing slight catarrh, while the stretching of the urethra behind the stricture leads to a slight chronic inflammation of the follicles of the mucous membrane in that part. We have previously learnt that this man has two strictures, one of large calibre one and a half inches from the meatus, and a second tight one four and a half inches down. In attempting to pass a bougie through this deep stricture, which ten days ago was found to be impassable, I might begin with an instrument of moderate size, say a No. 5, and, if I failed with that, take progressively smaller ones until I at last found one that would pass in. But it is a good point to reduce to a maximum all ineffectual attempts to pass an instrument ; they irritate the urethra, excite spasm, induce swelling of the mucous membrane, and so increase the difficulty. The better plan is at once to take a very small instrument that will probably pass, and so I will take this No. 1 whalebone bougie, and if it passes it will make it easier for me to pass No. 2. This is a striking fact in the treatment of stricture—we may express it in this way—failure to pass an instrument through a stricture increases the difficulty, while success with one instrument facilitates the passage of one still larger.

The grip on the bougie tells me that it is in the posterior of the two strictures. This gripping is a great hindrance to the further passage of the bougie, but, by exercising a little patience and care, I have now passed it quite through the stricture. I shall leave the bougie lying in the stricture till the end of my visit ; that is to say, for one or two hours, and we shall see then that the simple passage of the bougie lessens the constriction of the stricture. The mere presence of the bougie, whether it lies in tight or not, causes the stricture to yield. Some say that a natural absorption takes place ; some say that it is a yielding of the spasm. It is quite certain, however, that having once passed this in, by simply leaving it there we shall be able to pass afterwards a larger bougie.

Urinary Fistula.

This patient, a man of 25 years of age, has been sent in as a case of stricture. There is a history of gonorrhœa, and a catheter has been passed at intervals during the past eight months, apparently about six times in all. He states that he strains on micturition. In May, 1894, he had retention of urine for two days, and a medical man passed a catheter twice a day for some days. This occurred again later on in 1895, but then the retention only lasted one day. In May, 1894, a painful swelling appeared in the perineum, and was lanced a week after its appearance. The wound healed up six weeks after the operation. Since then there has been another swelling ; it also was lanced, and there has been ever since some yellow discharge from the opening. Occasionally the scrotum also swells. On examination we find an opening in the perineum, with indurated margins, and with swelling around, and the man states that when he passes water some of the urine escapes through this opening. It is important to know how much urine passes through this fistula. Some patients pass all their water through a fistula of this description, but here we see that only a few drops pass from the fistula, much the greater portion escaping from the meatus. The urine is slightly acid—a little turbid. It will be well for us to try to form an idea of the amount of pus it contains. Purulent urine is always turbid, even after prolonged standing, because *liquor puris* is a turbid fluid. The pus cells are to be seen with the micro-

scope. The chemical tests we use are generally two : if we add liquor potassæ, it becomes glutinous, and if we add some ozonic ether to purulent urine considerable effervescence occurs. The amount of pus will afford some clue to its source, whether from the perineal fistula or the kidney and bladder. In this case, the amount of pus is small, not more than might be afforded by the fistula. This urinary fistula is said to have followed upon the stricture which the man is said to have had, five years ago, after gonorrhœa. The question naturally arises, how these affections cause perineal abscess and fistula ? In the first place, the inflammation of gonorrhœa may extend to Cowper's glands and form a peri-urethral abscess, which, bursting externally, and into the urethra, forms an urethral fistula. This is a common way for a fistula to originate. Perineal abscess may arise in a case of stricture in the same way, the slight inflammation of the urethra at and behind the stricture may spread to Cowper's glands in a more acute form, just as we have seen that it sometimes extends to the epididymis. It is also taught that an hypertrophied bladder, capable of just squeezing urine through a tight stricture, will stretch the mucous membrane behind the stricture, and may make a tiny rent in it, through which a minute extravasation occurs, which sets up inflammation, ending in abscess. The patient says the lump in the perineum came after sitting on wet grass. It is well known that exposure to cold and wet—for instance, riding on a wet saddle, or sitting on wet grass—may give rise to inflammation, first by a process of congestion outside the urethra, and after that an abscess forms which burrows in certain directions, but has always a great tendency to burrow into the mucous canal, and form a urethral fistula.

I have already mentioned that this case has been sent in as one of extremely tight stricture ; we will now explore his urethra.

You notice that a No. 8 bougie passes with ease into the bladder ; a No. 22 French bougie in like manner also passes in with perfect ease, and therefore in the ordinary sense of the term there is no stricture to deal with now, and if there ever was, the treatment has been singularly successful in overcoming it. But the urinary fistula is still there. How shall we close this fistula ? It does not heal because of the constant irritation of the urine passing over it, and the object of our treat-

ment must be to prevent the urine thus flowing along the fistula. One way of doing this is to keep a catheter fixed in the urethra, so that all the urine will pass out through it. It is, however, quite unnecessary to have a catheter continuously lying in the urethra in order to convey urine which is passed, say, five times in the twenty-four hours, each act of micturition lasting two minutes only. The best way is to have a carefully prepared aseptic catheter passed every time the patient desires to pass water. The superiority of this plan over the other depends upon two circumstances. You remember that I have just told you that the whalebone bougie you have seen me pass with difficulty through the tight stricture of our first patient will in a short time become loose; just the same happens with a catheter. Even a full-sized catheter which just fits the urethra, if left in hour after hour, becomes loose. Urine then passes by the side of the catheter as well as through it, and escapes along the fistula and keeps it open. In the second place, you have not a foreign body lying hour after hour in the neck of the bladder, which may irritate the prostate, testicles, or bladder. This, then, is the plan I shall adopt with this patient: he will be taught to pass a catheter on himself, and told to use it whenever he desires to pass water, and also before going to stool, and I believe you will see that by this treatment alone the discharge from the fistula will speedily lessen, and after a time cease altogether. If for any reason this result is not obtained, I shall lay open the fistula and carefully follow up any lateral tracks I may find. I do not anticipate that this will be necessary.

Colotomy.

This is the patient upon whom I performed colotomy twelve days ago. The man has carcinoma of the rectum, and the operation was done for the arrest of haemorrhage and not for the relief of obstruction. You will remember that at the operation I found there was no mesocolon, and I could not put in the usual mesenteric stitch to bring the back of the bowel flush with the skin. Having fixed the ends of the loop of bowel to the skin by the two "fixation sutures," I sutured the skin to the bowel as far back as possible.

A week ago I cut away all the front and the sides of the loop of sigmoid flexure, and I want

you to see the result I have obtained. My object in doing the operation was to prevent the stool from going over the diseased rectum, and it is not enough simply to get a faecal fistula, but such a ridge or "spur" between the upper and lower openings of the colon that the faeces coming down will all pass out through the fistula, and none of it pass into the bowel below. Wherever possible, you secure this by the mesenteric stitch, which brings the back of the bowel level with the skin, but I want you to notice that in the cases in which there is no mesocolon—and they are not few—you can still obtain a thoroughly reliable "spur," if you are careful to pass your sutures well back in the bowel—as far back as you can place them. As the formation of an efficient "spur" is one of the chief advantages of inguinal-colotomy over the lumbar operation, it is important to know that we can secure it, even in the absence of a mesocolon. If an efficient "spur" is not obtained, and faecal matter passes down into the rectum, it accumulates there, and not only irritates the growth mechanically by the straining efforts of the bowel to pass it on, and by the hard masses grinding down through the stricture, but the chemical changes taking place in it may generate irritating compounds. However this may be, you will find that the beneficial effect of colotomy very largely depends upon the complete efficiency of the "spur" at the artificial anus, and the entire diversion of the motion. As I have said, it is because in inguinal colotomy you can produce a ridge which will prevent any of the faeces passing down to the rectum that you give the rectum more rest than you can by lumbar colotomy. You noticed, too, that I did not pass any sutures through the parietal peritoneum, but simply through the skin and the bowel. This method is not only much quicker and simpler than the older method, but I believe the union between the bowel and the belly wall is more secure and complete. If you put the stitches between the peritoneum and the skin, there may be slipping of the bowel adherent to the peritoneum from the skin. What you want is continuity between the mucous lining of the bowel and the skin, and you get it at once by stitching the bowel to the skin. Now as to what this man is to do. He will wear a belt; not one of the vulcanite hollow pads, but just simply a webbing belt six inches deep, fastened with three

straps and buckles. Lanoline is a good ointment to lubricate the part, outside which he will place a handful of cotton wool or a Gamgee sponge, and then a larger external pad of wool, and over all his belt. He will be able to do his work all day. At night he will take the pad off and wash the anus, and wear a lighter linen belt. In addition, it is useful to give these patients a charcoal biscuit after each meal ; it deodorises the faeces. Salol in 10 gr. doses in a cachet is also good for this purpose.

A CLINICAL LECTURE ON LYMPHATIC CONJUNCTIVITIS.

Delivered at the Victoria Hospital for Children

BY
W. T. HOLMES SPICER, M.B., F.R.C.S.

Lymphatic Conjunctivitis.

THIS disease appears in a great variety of forms, each of which has different stages ; all the forms, and some stages even, have received separate names, the result of which has been to unnecessarily complicate the understanding of one of the most common affections to which the eyes of children are liable. The terms pustular and phlyctenular conjunctivitis, exanthematous or eczematous conjunctivitis, pustular and phlyctenular keratitis, marginal keratitis, herpes of the cornea, scrofulous or strumous ophthalmia, are constantly used for this one and the same disease, the general type of which consists in the formation of a limited exudation of lymphoid cells on the surface of the cornea or conjunctiva, which breaks down by the removal of the covering epithelium, leaving a small shallow ulcer. There is no actual difference between the lymphoid elevations, whether they appear on the cornea or conjunctiva. They frequently exist in the same eye on the cornea and the conjunctiva at the same time, so that no useful end is served in making a distinction between these various forms of the same disease because they appear in different parts of the eye. It should

always be borne in mind that anatomically the anterior layer of the cornea is a continuation of the conjunctiva, and if the disease attacks the cornea, its seat is primarily the conjunctival layer of the cornea.

It is not an uncommon experience of the surgeon to have a child brought to him for advice on account of a speck which is said to be growing over the eye. Examination reveals the presence of a small grey opacity in the clear cornea, the result of a past attack of inflammation, the importance of which has not been sufficiently realised by the parents. Perfect transparency of the dioptric media of the eye is essential for complete acuteness of vision ; and the existence of a blemish on the cornea, if it be near the centre, means for the possessor of it a degree of disability for a period of months or years according to its depth and permanence. If the cornea has been seriously involved, complete restoration of transparency is not possible, and this may entail lifelong consequences. A person so affected may be incapable of serving in the army or navy, he may be unfitted for occupations requiring great delicacy of perception, he is constantly hindered by the loss of power of acquiring knowledge. His success in school life is marred, with the result of an enforced adoption of some less desirable occupation than that for which he would otherwise have been fitted. Above all, he may have diminished appreciation of the true form and beauty of external things, and a consequent loss of a great part of the pleasure of life.

All inflammatory processes in the cornea have as a result the production of a loss of transparency ; among other things this may be due to an ulceration and loss of the surface, or to an oedema or exudation into the proper corneal substance, producing a change in the relation of the corneal elements to each other, or their replacement by opaque fibrous tissue. Of all the ocular affections of childhood, by far the most common, the most tedious, the most difficult to treat, in some of its forms, is the group to which the best title that can be given is perhaps *lymphatic conjunctivitis*. The old term scrofulous or strumous ophthalmia, besides including all the eye affections of this group, was applied to all cases indiscriminately in which there was extreme intolerance of light. It was thought that the photophobia was induced by an irritation

of the optic nerve and retina and a consequent reflex spasm of the orbicularis. This view was incorrect, inasmuch as treatment, in the form of soothing applications to the terminations of the fifth nerve in the cornea and conjunctiva, lead to a diminution or disappearance of the intolerance of light; this intolerance also is not greater than that produced by the irritation of a foreign body under the upper lid. It is characteristic of all cases of superficial inflammation of the cornea in the growing period of life, that irritation, intolerance of light, and spasm of the lids are much greater than in the adult period. Nevertheless this classification under the heading scrofulous ophthalmia has this amount of truth in it, that in a large number of cases of superficial keratitis in the young there is a certain condition of the tissues which makes them favourably disposed to the occurrence of inflammatory outbreaks which are very chronic in their course and which tend to recur without obvious cause. This condition is associated with a proneness of the lymphatic tissue of the body to hypertrophy, whereby the lymphatic glands in the neck or at the angle of the jaw become enlarged, the mucous membrane of the nose becomes swollen and inflamed, and masses of adenoid tissue form in the pharynx; also if the family history and antecedents of these cases be carefully examined, it will be found that there is a great liability to the occurrence of tubercular disease in others of the family, or in their ancestors. I am aware that evidence of this sort has to be received with great caution, as it depends on the statement of friends who are not always well informed; but making all allowances for this, the association with a reliable history of tubercle will be found to be more marked than in persons who come under notice for other reasons. The liability to recurrence of this affection, its association with other lymphatic hypertrophies in the body, the frequent appearance of it in others of the same family, its association with tubercular affections, point to a peculiarity in the anatomical structure of the lymphatic elements of the body capable of being transmitted from one generation to another, and favourable to the growth of certain specific organisms. The actual relationship with tubercle is probably no nearer than this; the exudations in lymphatic conjunctivitis have been examined bacteriologically, and no tubercle bacillus has been found. The dis-

covery of the proximal cause of lymphatic conjunctivitis has yet to be made.

In its simplest form lymphatic conjunctivitis shows itself by the eruption of a single localized elevation on the ocular conjunctiva, ushered in by pricking pain, lacrymation, spasm of the eyelids, and avoidance of light. The elevation is at first covered with epithelium, and consists of a plentiful collection of lymphoid cells in the subconjunctival tissue; the conjunctival vessels around may be recognised by their mobility with the conjunctiva, their bright red colour, and their capability of being separately distinguished. The surface epithelium is shed during the course of a few days, and the swelling has a crater-like depression in its centre. Gradually the elevation flattens down, the epithelium creeps over the surface of the ulcer, and complete repair takes place. In more severe cases the elevations are multiple, and occur at the corneal limbus; when they are very minute they are known as *miliary phlyctenulae*. These are sometimes so numerous as to surround the whole limbus, or even to be scattered over the greater part of the surface of the cornea, which then has the appearance of being covered with minute grains of sand. The irritative symptoms are more severe, the conjunctiva of the lid is much swollen, there is most intense fear of the light, the child shrinks away to the darkest corner, or buries his face in anything that will shut out the light. At the slightest attempt to open the eyes a copious gush of tears comes out, and it is well-nigh impossible to obtain a view of the cornea. These confluent phlyctenulae produce a swelling of the limbus, while the cornea in the immediate neighbourhood is seen to lose its transparency, and contain a number of small grey exudations just beneath the epithelium. With the small phlyctenulae, as a rule, resolution takes place, the elevations and small corneal exudations disappear; in other cases the corneal exudation increases, so that small elevations are produced on the surface which lose their epithelium, and a crater-like ulcer is produced with a suppurating base. As a rule this ulcer heals; the manner of its healing is interesting: a number of vessels grow into the cornea from the limbus, surround the ulcer, and gradually creep over the ulcerated area at the same time that the epithelium grows over the surface and completes the process of repair. Sometimes

the ulcer deepens, and the exudation into the corneal substance can be seen extending as a distinct yellow haze all round the base of the ulcer. The iris is extremely congested and altered in colour; its actual blood-vessels can often be seen enlarged, and a formation of lymph or puro-lymph takes place in the anterior chamber. By gradual necrosis of the base of the ulcer, a limited perforation of the cornea results, with its consequences, such as prolapse of the iris, adhesion of the iris to the back of the cornea, or partial bulging of the cornea. This yielding of the cornea is generally attended by a sudden complete relief of pain, and the onset of the process of repair. In other cases the course is quite different: a number of vessels grow into the cornea from the limbus towards the crater-like depression, but instead of healing the ulcer advances slowly towards the centre of the cornea, carrying the leash of vessels with it; this produces what is known as *fascicular keratitis*. The advancing edge of the crater is convex towards the centre of the cornea, and somewhat raised in the form of a crescent, consisting of an exudation into the corneal substance, the vessels of the fasciculus ending in the concavity of the crescent. The difference between this fascicular keratitis and the natural healing of an ulcer by the process of vascularization lies in the fact that the crescentic advancing ulcer contains a yellow focus of purulent exudation. This condition may last many weeks or months, and is attended by great spasm of the lids, watering on exposure to light, and the formation of a band-like opacity, which remains visible for years.

Where the ulcers of the cornea are multiple and adjacent to one another, the ingrowth of vessels of repair from the limbus may occupy a considerable area of the cornea. These vessels remain as potential blood-channels for a very long time after the complete healing of the ulcer, and are liable to become easily congested, giving rise to a condition known as *phlyctenular pannus*, or *superficial vascular keratitis*. These vessels lie in the sub-epithelial part of the cornea, and sometimes cover a considerable area; they are at times invisible except to careful examination, and at others, during the active stage of a recurrence, are much enlarged. If one considers all these forms of the affection, it will be seen that although the clinical appearances of lymphatic conjunctivitis are extremely varied,

they are all alike in their origin and in their tendency to recur, but vary much in their tendency to repair or to penetrate deeply into the cornea. As a rule, the marginal exudation tends to heal with a more considerable ingrowth of vessels than the central parts, and for this reason, possibly, to leave less permanent or marked results in the cornea.

Causation.—This is a disease of childhood, but it does not occur generally in children under one year old, its most common period is from two to sixteen years; during this age skin eruptions, eczema, and impetigo about the face are very common, together with a similar condition of the nasal mucous membrane; possibly direct infection of the cornea may take place in some cases from one of these sources. It most commonly occurs when the changes in temperature are great and sudden, as in spring and autumn. As the effect of these climatic changes is greater among people who live in damp, ill-ventilated or overcrowded houses, so we see that this disease is common among the poorer classes, and rarely occurs in children whose hygienic environment is quite satisfactory. As a probable result also of the environment, children fall into irregular habits of feeding, and eat anything at any odd time, instead of having regular meals, whereby the appetite becomes capricious, and the desire for unwholesome food becomes established.

The next most common exciting cause is a previous attack of some acute exanthematous disease, such as measles, scarlet fever, whooping cough, &c., by far the most common of which is measles. The eye affection comes on a few weeks after the measles, and must not be confounded with the conjunctival congestion which commonly ushers in the first stage of measles. The irritation of head lice is a frequent accompaniment of the disease, and is more likely to occur in such children, as during this period they appear sufficiently morose and unattractive to make neglect probable.

Treatment.—The slighter cases as a rule get well if left alone; a little weak antiseptic lotion, such as a solution of boracic acid, ten grains to the ounce, should be dropped into the eye twice or thrice a day, and a small piece of yellow oxide of mercury ointment of the strength of five to eight grains to the ounce should be introduced between

the lids by means of a small camel's hair brush, night and morning ; gentle friction should then be applied to the lids to insure the distribution of the ointment all over the conjunctival sac. Other remedies equally good are zinco-cyanide of mercury ointment, half grain to one grain to the ounce, or the application of calomel in powder to the everted lower lid. The action of the calomel is less irritating than that of yellow oxide ; it is better to use it, therefore, in the earlier stages, when the healing process is not thoroughly established. The calomel should be pure and quite dry, otherwise the intensely irritating corrosive sublimate forms in excess, with the result of increasing the irritation. Its action no doubt is a chemical one, probably by means of the conversion of the subchloride into the perchloride by the action of the salt in the tears. A caution is necessary here : the administration of iodine in any form internally is contraindicated while calomel is being used locally. Otherwise the very corrosive green iodide of mercury is formed. I have twice in the past year seen an ulceration of the conjunctiva of the lower lid with a green deposit in it, as the result of the local application of calomel, while the patient was taking iodide of iron internally. If the case is more severe, with intense spasm of the eyelid and much avoidance of the light, great care is needed in obtaining a satisfactory view of the cornea. In dealing with a child affected in this way it is necessary to gain its confidence, so that it feels that it will not be unnecessarily hurt. The first instinct of the mother is to seize the child and forcibly hold up the lid, with the result of increasing the spasm and ruffling the temper of the patient ; by gentle encouragement a sufficient view of the cornea can often be obtained by turning the child away from the direct light, and urging it to open the eye while gently lifting the upper lid. It is not often necessary to use mechanical aids for the forcible opening of the lids. In most cases the two thumbs are enough ; occasionally it is necessary to be brutal and force open the lids by means of the lid retractor ; this occurs when the veins of the lid are much enlarged by the moist warm state in which the eyes are kept by the knuckle or pillow being thrust into the eyes to exclude the light. The veins of the lid pass between the fibres of the obicularis, and are kept in a state of congestion by the firm contraction of the fibres ;

in such a case, on trying to open the eye, the lid is everted, and fold after fold of deeply engorged conjunctiva comes into view completely covering the cornea. The difficulty is increased where there are excoriations of the lids at the commissure, a condition known as cracked canthi. The best method of making an examination is for the child to lie on the nurse's or mother's knee, its head being received between the knees of the doctor, who is sitting opposite. There should be at hand, within easy reach, a bowl of cold water, a few small sponges of absorbent wool wrung dry, a camel's - hair brush, some solution of nitrate of silver and of atropine, so that the remedies may be applied at the same time that the examination is made. It is occasionally necessary even to give the child an anæsthetic to examine the eye. Whatever means be necessary, the cornea should be seen at any rate at the first examination, so that the surgeon may know with what degree of severity of the affection he has to deal. Sometimes where the spasm of the lid is greatest, the actual affection of the cornea is very slight indeed, and is confined to the surface only. It is often possible to obtain relief from this spasm by allowing a stream of cold water to fall on to the eye from some height ; this should be done two or three times a day as routine treatment as long as the spasm lasts. Where the spasm is greatest, I have found it most useful to dry the conjunctiva as far as possible, and to apply freely to it and to the skin of the lid, especially if eczema is present, a solution of nitrate of silver, ten grains to the ounce, mopping up the excess. By this means the overloaded vessels empty themselves ; the surface layer of the conjunctiva is covered with an extremely delicate layer of slough, which appears as a white bloom on it, in which the irritated nerve endings are involved. A few hours after the application, the child may spontaneously open the eyes.

If the cornea be involved either by an ulcer or by an exudation into its substance we should abstain from all irritative treatment like yellow oxide of mercury or calomel. Atropine should be used either in the form of drops or ointment in the strength of 2 or 4 grains to the ounce. By using atropine in the form of ointment there is less risk of producing toxic symptoms from its absorption, such as thirst, dryness of the throat, and delirium. The atropine remains in contact with

the eye for a longer time, and its local absorption and effect on the pupil are correspondingly greater. Atropine should be used sufficiently often to maintain full dilatation of the pupil ; it should not be smeared freely over the lids, as is thought necessary by parents. If used too often or too freely it gives rise to a highly congested state of the skin of the lid and face, and of the conjunctiva, a condition known as *atropine irritation*. In some persons who are very susceptible to its action, atropine causes this peculiar irritation apart from too free use of it. Whenever atropine has been used, and much redness of the skin comes on, this toxic effect should be borne in mind ; otherwise it may be thought that the ulcer is worse and needs more atropine. The drug should be at once discontinued, and boracic acid ointment should be smeared over the irritated skin. When the ulcer is very deep the posterior elastic layer of the cornea, Descemet's membrane, may be seen bulging at the bottom of the depression as a small black projection. Where this condition is present it is better to get the pupil quickly under the influence of eserine, 1 grain to the ounce ; this diminishes the risk of a serious prolapse of the iris. At the same time the eye should be bandaged, and if painful should have hot fomentations. If the ulcer shows no tendency to heal under this treatment, it is better to puncture its base and evacuate the aqueous while the eye is under the influence of the eserine.

After perforation the change in the condition is sometimes very striking—the pain ceases, the patient obtains sleep, and the healing process starts as a rapid ingrowth of vessels to the ulcer. The experience of some is favourable to the use of eserine as routine treatment in corneal ulcers of this kind, apart from the risk of perforation, in the strength of $\frac{1}{2}$ grain to the ounce to be used every three or four hours ; it is said to promote healing. I cannot speak from my own experience with very great favour of this form of treatment ; as a rule I have found it less successful than atropine, but I have no hesitation in using it where atropine fails ; in those cases of central ulceration with no tendency to vascularization where the nutrition of the cornea is at fault, I believe it to be a very valuable agent. Eserine produces contraction of the pupil, dilatation of the vessels of the iris and of the ciliary region, influences the nutrition of the cornea by pro-

moting the vascularity of the region from which it draws its nutritive supply. Sometimes an entire change of treatment, from eserine to atropine or *vice versa*, is in itself beneficial, and I have sometimes seen good results from one form of treatment in one attack in the same patient in whom an apparently similar attack had been previously successfully treated in the opposite way.

The fascicular form is very tedious. If the crescentic area is yellow and infiltrated or certainly advancing, it is necessary to use other measures, such as curetting or local cauterisation of the ulcer. The inflamed area should be scraped with a sharp spoon, so as to remove the infiltrated tissues, or lightly touched with the galvano-cautery, or with a sharp stick of mitigated nitrate of silver, carefully neutralised afterwards with chloride of sodium. Section of the fasciculus does not stop its advance. Bandaging the eyes, as a rule, is not advisable ; coolness, free access of air, and the non-avoidance of light are the best for the patient. The bandage is only necessary where there is risk of perforation of the cornea. To protect the eyes from the glare of bright daylight goggles may be used, or a shade to cover both eyes ; bleeding and blisters are useless. I have found the use of a seton in the scalp sometimes very active in promoting permanent cures of the most obstinate cases where all other treatment had failed. If it is used it should be kept in place till the bridge of skin is worn through or until after every trace of irritation in the eye has disappeared. This form of treatment is barbarous and unpleasant, and may lead to serious cellulitis of the scalp with suppuration of the neighbouring glands, so that I have for a long time ceased to use it, and I do not find many cases which obstinately resist all other kinds of treatment. Two difficulties frequently present themselves to one's mind in dealing with these ulcers, and may be considered here. One is to decide whether the ulcer is in a progressive or quiescent stage ; it is safe to assume that in this form of ulcer, if there be no fear of light and no congestion of the vessels of the circumcorneal region, the ulcer is quiescent. To make sure of this a drop of fluorescin solution should be inserted just inside the lower lid, and after about half a minute one or two drops of cocaine or boracic acid should be dropped into the eye ; if there be loss of the corneal epithelium the

denuded area will be stained bright green ; if the epithelium be intact or healed it will not be stained.

The other question is how long should the treatment of the corneal ulcer be continued ; the effects of the ulcer on the cornea vary with its duration and depth ; after the acute irritative stage is passed, dilute yellow oxide of mercury should be used till the resulting opacity has either disappeared entirely or ceased to change, which can only be told by observation, or until the facets left in the cornea have been completely filled up, and the surface again presents an uniform level. Such treatment may have to be carried out for many weeks or months.

As to general treatment, it is well to begin in the earlier stage with a purge ; this should be followed by iron, iodine, arsenic or quinine, together with the administration of cod-liver oil. The child should be made to go out in the fresh air as much as possible, regardless of the intolerance of light. A change of air and diet is frequently one of the most valuable aids, regular food at definite intervals should be insisted on, and constant recourse to cakes or unripe fruit should be prevented. There seems to be something in the digestive conditions of these children that makes them devoted to unripe fruit. For this reason, wherever it is possible, a change of surroundings is most salutary, even if it be the removal to the ward of a hospital, so that the diet may be properly regulated. This is one of the most important elements in the treatment. It is frequently enough only to place the child in a well-ventilated hospital ward, away from the damp and crowded home, to ensure rapid recovery ; if it can be arranged, a change of scene into the country, or to the seaside—at some bracing place like Margate—is most useful as a generally invigorating measure. The treatment of blepharitis and eczema of the face and scalp should be carried out thoroughly at the same time. Ammoniated mercury ointment or dilute nitrate of mercury ointment with oxide of zinc should be applied after the removal of all crusts by warm alkaline lotions. The treatment of the nasal passages is most important : the removal of adenoid growths, the disinfection of the pharynx, and the re-establishment of the permeability of the nasal air-passages and the suppression of the discharge from the mucous membrane should be

taken in hand promptly. The effect of this is almost certainly not local, that is by means of a communication through the nasal duct, but by reason of the benefit to the patient's condition brought about by the removal of a source of constant irritation from the naso-pharynx and the re-establishment of free access of air to the lungs.

REVIEW.

THE "LANCET" AND THE HYDERABAD COMMISSIONS ON CHLOROFORM.

Being the Report of the "Lancet" Commission appointed to investigate the subject of the administration of chloroform and other anaesthetics from a clinical standpoint, together with the reports of the First and Second Hyderabad Chloroform Commissions.

THE perusal of the introduction to this volume tells us that the main factors in initiating the Hyderabad and "Lancet" Commissions were discrepancies between the death-rates under chloroform in India and, perhaps it may be added, Scotland, and that observed in other parts of the world. A matter of daily observation, at all events in Great Britain, is the comparative frequency of deaths under chloroform ; on the other hand, many thousands of successful cases of operation under that anaesthetic without a single death were recorded by Lawrie in India. It was this distinguished practitioner who, following the teaching of his master Syme, rather than of Simpson, attributed the deaths to imperfect knowledge and faulty method of administration. He pointed to Syme's practice and his own as being based upon what he holds to be the only safe way of giving chloroform as a proof that death does not follow if certain simple rules are carried out in their entirety. In order to prove his point, he undertook, in conjunction with various surgeons practising in India, a series of experiments on the lower animals, which were published under the designation of the "First Hyderabad Chloroform Commission," and which appears in the present volume as an appendix on p. 78. These experiments were divided into four series :

1. Eight animals were anaesthetised with chloroform until death took place.
2. Seventy-five experiments were made, large doses of chloroform being used, and the effects of artificial respiration were carefully watched.

3. Seventeen experiments upon animals anaesthetised with drachm doses of chloroform with and without artificial respiration.

4. Forty-one experiments were made upon animals, which were rapidly poisoned with large doses of concentrated chloroform vapour given as far as possible without admixture of air.

The results in the first series were roughly :—(1) Partial loss of sensation with slightly increased reflex irritability, followed by (2) complete loss of sensation and of reflex action ; (3) cessation of respiration ; (4) absence of pulse in the femoral artery ; and finally (5) stoppage of heart's action. "Although the intervals of time between the occurrence of these events varied within wide limits in different cases, in no instance was the sequence modified." "The heart's action never became dangerously affected or stopped until after cessation of respiration." The deductions from the second series of experiments were thus summarized :—(1) In no case did arrest of the cardiac action from syncope take place during the first stage. (2) In no case was there reflex inhibition of the heart's action. (3) The inhalation of chloroform vapour, however given, cannot kill a dog by direct action on its heart. (4) "The Commission further consider that chloroform vapour administered to dogs never kills by acting on the intra-cardiac ganglia either primarily or secondarily. It is impossible to produce syncope from chloroform in dogs." From the third series, although the results were, in the main, the same, the following points were further noticed :—The heart ceased to beat in very slow chloroformization (two and a quarter minutes) more rapidly than when the vapour was given more quickly. Artificial respiration was tried in five cases, the heart sounds being barely audible. One animal revived. The fourth series illustrated the following points :—(1) Anaesthesia was produced very rapidly (one minute and a quarter) when large doses of chloroform were given. (2) The heart's action was said to be less hampered in these cases than when anaesthesia was produced more gradually ; artificial respiration tried in one of these cases was successful, after the pulse had ceased for twenty-eight seconds. The Commission came to the conclusion that there was little tendency in dogs towards the accumulative effects of chloro-

The more concentrated the vapour, the

more rapid the anaesthesia, unless an overdose interfering with the respiratory centre was given. They believed that the elimination of the drug was also more rapid. In no case did cardiac syncope occur.

These conclusions, which may be summed up by saying that chloroform kills wholly and solely by paralysis of respiration and never through cardiac syncope, were put forward in the pages of the "*Lancet*." It was, however, pointed out that the number of the experiments, as well as the conditions under which they were carried out, did not render it possible that they should be accepted as final. Dr. Lawrie therefore obtained the assistance of His Highness of Nizam, and through the generosity of that potentate arranged a second Hyderabad Chloroform Commission. This consisted of eminent surgeons in India, assisted by Dr. Lauder Brunton, who went out as the representative of the "*Lancet*." The work of this Commission is published in the volume before us, and was of a much more thorough, precise, and complete nature than its predecessor. The experiments were 588 in number, and were in many cases "recorded," the records being photographed and printed. This is a point of great importance ; the actual unalterable tracings can be seen and studied in the volume before us. The experiments covered the ground of the first Commission, and further were conditioned as far as possible in such a way as to test the action of fatty degeneration, surgical shock, and posture upon the chloroformed animal. How far the conditions bear the test of criticism we shall have to consider later. The conclusions deduced from the experiments are thus summarized :—Chloroform, when given continuously by any means which ensures its free dilution with air, causes a gradual fall in the main blood pressure, provided respiration is unimpeded ; the fall continues, the animal becoming first insensible, respiration then fails, and finally the heart stops. If the chloroform is less diluted the fall is more rapid, and is always gradual, however concentrated the vapour. Sudden deaths from stoppage of the heart never occur. The greater the degree of dilution the less rapid the fall, until the degree of dilution is reached which no longer appreciably lowers blood pressure or produces anaesthesia. If inhalation is interrupted at any stage, the fall in pressure continues. This is

probably due to the absorption of the residuum of chloroform in the air passages. If the chloroform is stopped in the early stage, pressure soon begins to rise; if chloroform is pushed further to a point which is not easy to define, blood pressure and respiration will no longer be restored spontaneously, although the heart continues to beat. If the fall has been very gradual, respiration sometimes stops completely till blood pressure rises again, the respiration recommencing spontaneously in the course of the rise. This may occur after the inhalation has been discontinued; as a rule, "If respiration has stopped or even become slow and feeble at the time when the inhalation is discontinued, and artificial respiration is not resorted to, fall in blood pressure will continue until death ensues." Struggling and holding the breath cause variations in the gradual fall of blood pressure; the former causes a rise in blood pressure independent of any change in respiratory rhythm. When accompanied by acceleration of respiration and pulse, especially if the breathing be deep and gasping, more rapid intake of chloroform occurs with acceleration of fall of blood pressure, and great after-fall. Involuntary holding of breath leads to rapid fall of blood pressure, with slowing of the heart's action; on resumption of breathing, the pressure rapidly rises, gasping respiration occurs, increased intake of chloroform, rapid insensibility, fall of blood pressure and onset of dangerous symptoms—"The combination of struggling with alternate holding of breath and gasping, which results if the chloroform is applied closely to the face without sufficient dilution with air, causes violent fluctuation, and then a speedy fall of blood pressure, which however soon leads to dangerous depression with deep insensibility, and early stoppage of respiration. The after-fall under these circumstances is rapid and prolonged." Slight continued asphyxia, from whatever cause, causes irregular and exaggerated remission of the blood pressure with slow and irregular heart's action. Commonly associated with it is gasping expiration leading to increased intake, and as a consequence rapid decline of blood pressure.

Complete or almost complete asphyxia produces similar but more marked effect—"This effect of asphyxia is the result of stimulation of the vagi, the proof of this is—

(a) That the trace corresponds exactly as stated

above to that produced by direct irritation of the vagus.

- (b) Division of both vagi entirely abolishes it.
- (c) The administration of atropine which paralyses the vagus also abolishes it."

This slowing or temporary stopping of the heart, with great fall of pressure caused by vagal irritation is, the Commission states, protective rather than perilous; less blood containing chloroform is pumped throughout the circulation. Upon the other hand, it appears to us that even if this hypothesis be correct, an important fact is lost sight of, for not only have we to deal with the chloroform entering the blood from the tidal air, and so being conveyed to the nervous centres, but also with the chloroform already in the circulation, and in the tissues perfused; in other words, the chloroform which is being intaken, and the chloroform which is already acting upon the tissues. Now, unless *pari passu* with the cessation of the circulation of chloroform-laden blood we can insure the due elimination of the chloroform already in the blood, we are not placing the animal in a position of safety. It may be perfectly true that the enfeeblement of the circulation may protect the organism by conveying less fresh chloroform into the tissues, but equally will it fail in deporting from the tissues the chloroform which had already reached them. This objection does not appear to have been met, so far as we can gather from the report before us. The question of the safe period which exists between cessation of respiration and restoration of breathing by artificial means is dealt with, and it is shown that artificial respiration fails unless commenced very soon after the failure of natural breathing. Chloroform injected into the heart through the jugular did not, it was found, cause clotting. A large number of drugs were given before the exhibition of chloroform, morphia for example was regarded as a synergist, as it appeared to narcotize the respiratory centre, although it is noted that it had no "effect in shortening the period that may be allowed to elapse between the cessation of natural respiration and the commencement of artificial respiration." The other drugs used had no effect upon the action of chloroform except when their own special action became the leading feature in the case, as, for instance, the vomiting from apomorphine, or the violence of the convulsions produced by nicotine.

A number of experiments were undertaken to test the alleged danger from shock during chloroformisation, whether complete or imperfect: "In such cases a slight variation in the blood pressure would sometimes occur, such as one would expect from the irritation of a sensory nerve, or from the struggling that ensued, but in no case in any stage of anaesthesia was there anything suggestive of syncope or failure of the heart's action." And it is added, "If chloroform really had any power to increase the tendency to shock in operations, it is impossible to believe that it would not have been manifested to some degree at least in one or other of these numerous experiments." Another kind of experiment was tried: "An animal that was put in a condition of extreme danger (from which it could only be restored by means of artificial respiration), by inhalation of chloroform for one minute, recovered readily and spontaneously after five minutes of chloroform inhalation together with inhibition of the heart by electrical irritation of the vagus carried on simultaneously." It is further noted that artificial respiration may place the animal in extreme danger by pumping the chloroform already in the lungs into the circulation; but here again it appears to have been overlooked that while chloroform is pumped in it is also pumped out, nor does it seem to have been made clear under what circumstances artificial respiration can be regarded as a safe means of recovery to a patient from an overdose of chloroform. The Commission concludes: "Chloroform has no power of increasing the tendency to either shock or syncope during operations. If shock or syncope from any cause does occur, it prevents rather than aggravates the dangers of chloroform inhalation." Then follow numerous experiments contrived to test the effect of fatty degeneration of the heart substance. Animals fed with phosphorus, and dieted in particular ways, were given chloroform, but only negative results were arrived at. The Commission, indeed, appears to have placed little reliance upon these experiments, and probably justly thinks the conditions were very far removed from those presented in clinical work, although they say, "The truth about fatty heart appears to be that chloroform *per se* in no way endangers such a heart, but on the contrary by lowering the blood pressure lessens the work that the heart has to perform, which is a positive advantage."

An important addendum follows. "A patient with an extremely fatty heart may die from the mere exertion of getting on the operating table, just as he may die in mounting the steps in front of his own hall door, or from fright at the mere idea of having chloroform, or of undergoing an operation or during his involuntary struggles. Such patients must evidently die occasionally during chloroform administration, and would do so even were attar of roses or any other harmless vapour substituted for chloroform." In like manner, extreme haemorrhage is said to be protective as producing a less rapid intake of chloroform. Posture is not regarded by the Commission as of much importance. "Various operations were performed on animals in the vertical position, but in no case was anything resembling danger from shock produced." Inversion of the body produces increase of blood pressure in the carotid, but failed to restore an animal which was in the last stage of chloroform poisoning, although it raised the pressure as long as it was continued. This rise of blood pressure is regarded as purely an effect of gravity.

The experiments undertaken with ether were clearly unsatisfactory, as owing to the temperature and the apparatus employed, the animals were never properly etherized, and therefore we are not surprised when we find the Commission speaking of ether narcosis as "semi-anaesthesia," and wholly unfit for surgical procedure.

One hundred and eighty-six manometric experiments are noted, and are well worthy of close study, while the tracings reproduced greatly enhance the value of the work. One or two of these we note do not properly belong to the Commission, but still have their own value. The practical conclusions which bring to an end the second Hyderabad Chloroform Commission are very similar to those issued by the report of the Royal Medical and Chirurgical Society in 1864, and may briefly be summarised as insisting upon a recumbent posture with absolute freedom of respiration; this in spite of the Commission's experiments, which seem to prove that the vertical position, at all events for the lower animals, is equally safe. The patient's chest and abdomen should be exposed during chloroform inhalation; if the breathing is embarrassed, the lower jaw should be pushed or pulled forward so that the lower teeth protrude

in front of the upper, this raises the epiglottis and frees the larynx. If any accident occurs and respiration stops, artificial respiration is to be commenced at once, by the combined Sylvester and Howard's methods, and should be continued until there is no doubt of the re-establishment of natural respiration. A small dose of morphia may be injected subcutaneously before chloroform inhalation, as it helps to keep the patient in a state of anaesthesia in prolonged operations. Alcohol may be given with advantage before operation under chloroform. Minute directions are given with regard to respiration, and here, as throughout the report, emphasis is laid upon the necessity of giving undivided attention to the respiration, while all watching of the pulse or heart's action is deprecated.

Thus, with no uncertain note the two Hyderabad Commissions undertake to prove and have to their satisfaction established their point, that chloroform is an innocuous drug, when given freely diluted with air, and when the slightest change in respiratory rhythm is watched for and treated as the indication for lessening the strength of the vapour inhaled. An important hiatus, however, was felt to exist in the chain of proof, and to remedy this the whole question was opened *de novo* from the clinical side, and the "Lancet" appointed Dr. Dudley Buxton to collect statistics both of normal and fatal cases of chloroform inhalation and to compare as thoroughly as possible the facts resulting from his labours with those given in the Hyderabad reports. More than half of the volume before us is occupied with this report, which is of the most elaborate nature, and it is impossible to do it justice in our present issue; we may perhaps, however, anticipate so far as to say that the Hyderabad conclusions are not borne out by the result of the clinical inquiry. Certainly it would appear that in temperate climates death from circulatory failure is not an unknown or an unusual mode of death under chloroform, and if we may accept the evidence of the clinical report, we must refuse to regard as a safe rule that the patient's respiration, taken by itself, is a sufficient guide to his condition under chloroform. Perhaps one may even go a step further and say that the clinical inquiry brings out in a very plain way the necessity for careful watching of the circulation in a patient submitted

to chloroform. We, however, reserve the further consideration of the clinical report for another issue.

CASES DEMONSTRATED AT THE CLINICAL MUSEUM

BY

JONATHAN HUTCHINSON, LL.D., F.R.S.

Reported by J. T. CONNER, M.D.

Crateriform Ulcer on the Forehead and Epithelioma (Rodent Ulcer) on the Cheek.

THE subject was a feeble old woman, aged 81. According to her account, the sore on the forehead commenced five months ago, and that on the cheek soon afterwards. But the dates cannot be trusted, as her memory was evidently defective.

On the forehead, on the right side, was an ulcer as large as a watch face. It was circular, and had a somewhat elevated bossy edge, enclosing a large shallow excavation. The edge nowhere presented rolled outline of the rodent. The surface showed no granulations, and was covered with a grey film. There were no satellites, and the surrounding skin was but slightly congested. The sore on the left cheek was very different in character. It consisted of a group of somewhat indurated nodules, which were covered with a yellowish-grey, adherent epidermic crust. None of them were ulcerated, but one was florid and threatening to become so. The patch did not present the characters either of the rodent or the crateriform ulcer. There was no gland disease. The upper one was diagnosed as the crateriform ulcer, though it did not present nearly so deep a crater as is usually seen in that form of disease. The lower one was probably epitheliomatous, and approached the type of rodent.

The case illustrated the tendency of the senile skin to produce malignant growths at more points than one, of which Mr. Hutchinson had published many examples.

Verruca Plana.

The patient was a young woman, aged 21,

brought by Mr. T. J. Hitchens. The backs of the hands and the lower halves of the forearms were covered with small flat warts, all about the size of pins' heads, and of the same colour as the skin. They were both irregularly disseminated, and also here and there collected into closely-set groups, or arranged in lines. None were present on the face or elsewhere. They had been coming out for the last two years.

Mr. Hutchinson remarked on the great number but small size of the warts. They constituted the variety known as "Verruca plana." The pathogenesis was to be explained thus :

There was a tendency to overgrowth of papillæ (papillomatosis) at certain ages. When one wart was formed it was liable to give rise to others by a process of infection.

Syphilitic Lupus.

The patient was a widow, æt. 45, brought by Mr. Sequeira. The bridge of the nose was stippled with small scars. The skin of the upper lip, between the nose and mouth, was covered with a slightly elevated, red, but somewhat brownish growth. This was arranged in irregular lines, interspersed with scars, which, like that on the nose, were perfectly sound. At a little distance to the left was an adherent purulent crust. On the left cheek, near the nose, was a small ulcer; and on the right, but further out, was another, with an oedematous edge. There were a number of healthy scars below the right knee, such as would result from gummatæ. The history was as follows :— Soon after marriage, twenty-two years ago, she had a rash all over the body. Her husband suffered from sore throat for many years, and died at 37 from "bronchitis and dropsy." There were three children and no miscarriages. The eldest, now aged 20, attended the demonstrations on Nov. 27th, 1894, and showed interstitial keratitis and syphilitic teeth.

Mr. Hutchinson pointed out that, apart from the history which was so definite, the condition of the face could be diagnosed as syphilitic lupus and not lupus vulgaris on several grounds: there was no "apple jelly" deposit; the scars were quite healthy; in lupus vulgaris there would not be a number of distinctly separate scars but one continuous one, nor would ulcers occur at a distance from the patch.

An Eruption due to the Administration of Bromide of Potassium.

The patient was a boy, æt. 3 years, brought by Dr. Pugh. He had an attack of measles fourteen days ago, which was mild; and was given five grains of the bromide three times a day for a week. Eight days ago, and, according to the mother, six days after he had taken any medicine, the present eruption appeared. It consisted of papules varying in size from a pin's head to a pea. The larger ones were framboesoid, resembling redcurrants, and had a slight areola. The eruption was imperfectly symmetrical. It almost exempted the trunk, was sparsely distributed on the limbs, but thickly on the face, especially on the cheeks. On the right calf was an elevated fungating granulation mass covering an area equal to a florin and distinctly constricted at the base. The mother stated that some months ago there was a boil in this situation. It healed, leaving the skin red, and exactly on this spot the granulation mass had formed.

Mr. Hutchinson showed a number of portraits illustrating the bromide rash. He said that the growth on the calf, which differed so much from all the other lesions, was explained by the damage done by the previous inflammation providing a "locus minoris resistentiae."

REVIEW.

Public Health Laboratory Work. By HENRY R. KENWOOD, M.B. (Second Edition, Lewis Practical Series). Price 10s. 6d.

This manual has already gained for itself such an established position as a practical guide that the issue of a second edition is very welcome. The book is fully up-to-date in the processes suggested and forms a most reliable guide to the subjects treated of. The D.P.H. Diploma is now being much sought after by all, and we are frequently asked questions as to the best books to read for the purpose; we have no hesitation in saying that Dr. Kenwood's book is amongst those which must be enumerated as necessary, and we confidently expect that a fresh edition will soon be required.

THE CLINICAL JOURNAL.

WEDNESDAY, AUGUST 26, 1896.

A CLINICAL LECTURE ON CHRONIC BRIGHT'S DISEASE.

Delivered at Charing Cross Hospital, June 1, 1896, by
T. HENRY GREEN, M.D., F.R.C.P.,
Senior Physician to the Hospital, and Physician to the
Hospital for Consumption, Brompton.

GENTLEMEN.—You recognize this patient, a case of chronic Bright's disease, that we have had under observation in Victoria ward during the last three months. From first to last the case has been so full of clinical interest, that I propose to-day to review its most important features, and to emphasize some of the lessons they have taught us.

A dangerous deficiency in the excretion of urinary solids, a minimum of cardiac hypertrophy and increased blood-pressure, and the administration of pilocarpine during an unusually long period, have, as you will remember, been the main characteristics.

The patient, a married woman, 31 years of age; was admitted at the end of February last on account of an attack of convulsions, with coma, obviously uræmic, which occurred at the Ophthalmic Hospital, where she went on account of some defect of vision. A year previously, after the birth of her last child, she noticed that her sight was impaired, and she began to suffer from occasional headaches, sickness, and polyuria, being obliged to get up two or three times in the night to pass her water. Since then she has attended an eye hospital from time to time on account of her sight. We find that two and a half years ago, after the birth of her first child, she had a "fit," which was possibly uræmic; but with this exception her health appears to have been good until her last confinement a year ago. There is probably some causal relation between her renal disease and her pregnancies. Further, at the age of ten years she had scarlet fever. There is nothing else in her history worthy of note. At the present time she is so much better that she is able to be up

and to take solid food; and she will tell you that were it not for her "weakness" and eyes she would be pretty well.

Firstly, a few words about diagnosis. The recognition of the renal disease presents no difficulty. If she came to you now for the first time and told you that her sight was defective, and that she had suffered from headaches and sickness, you would naturally examine her urine.... This is pale in colour, of specific gravity 1.006 and contains one fourth albumin. Examination of the urine alone is therefore quite sufficient to enable us to say with certainty that she has disease of the kidneys; the low density with so much albumen could only result from organic renal disease. Then, if you examine her heart and circulation, as you would do in every case of Bright's disease, you find some enlargement of the left heart, accentuated aortic second sound, and a persistent pulse. In the second place, the disease is obviously not recent. The cardiac hypertrophy, low specific gravity of urine, and clinical history, place the case in the category of *chronic* Bright's disease. You will remember I have often insisted on the importance, in every case of Bright's disease, of endeavouring to determine at the outset whether the disease is recent or old. Speaking generally, it may be said that recent cases, those which, owing to a more or less acute onset, reveal themselves in the early stage, if properly treated, get well; the longer the duration of the disease, the worse the prospect of recovery. You cannot devote too much care and attention to the treatment of recent and recoverable disease. Want of care here is responsible for many of the chronic cases.

We endeavour, in the next place, to classify the case—to which variety of Bright's disease does it belong? The absence of any diminution in the quantity of urine, and of dropsy, together with the cardiac hypertrophy, make the existence of *granular* kidney certain, but whether the primary degenerative form or the result of a chronic nephritis, is I think doubtful. That an inflammatory process has played some part in the renal disorganisation would

seem probable from the association of the disease with pregnancy, its somewhat rapid progress, and the large amount of albumen. Here we are confronted with a difficulty, by no means uncommon, that of being able to refer every case of Bright's disease to its proper category. But we must remember that our classification is, after all, more or less artificial, and that so-called "mixed" cases are not infrequent.

Much more important than this attempt to classify, and indeed a necessary preliminary to it, is to estimate the extent to which the functions of the kidneys are impaired, to note the changes in the heart and circulation, and to see how far the general nutrition of the patient has suffered in consequence of the disease.

An examination of our patient's urine shows at once the serious extent to which the functions of the kidneys are impaired. The daily excretion of urinary solids (taking the last 24 hours urine) is only 19 grammes, the urea 12 grammes. This is only about one third of the normal. The estimation of solids is of the *first* importance in this inquiry; and for practical purposes the simple arithmetical method we are accustomed to in my wards, in which the solids are calculated from the specific gravity, is sufficiently accurate. All that is necessary is to measure in cub.-centim. the quantity of urine passed in the 24 hours, and its sp. gr. Then, last two figures of sp. gr. \times 2 \times $\frac{1}{1000}$

= grammes of solids in 24 hours. When greater accuracy is desirable, a quantitative analysis of the urea must be made by Liebig's or some other process, but in most cases the above simple and easy method is sufficient. I need scarcely warn you against drawing conclusions from a single examination. In all urinary investigations repeated examinations at sufficient intervals are necessary. These urine charts extend over a period of three months, and a glance at them will show that during the whole of this time there has been a grave deficiency in the excretion of urinary solids.

The albumen and casts do not help us much in this inquiry. They indicate the existence of the disease, its quiescent or progressive character; but they are of little value as measures of the extent to which the kidneys are already damaged and their functions impaired. The loss of such large quanti-

ties of albumen in our patient's urine is an important factor in the causation of her weakness.

We proceed in the next place to estimate the changes that have taken place in the heart and circulation. Here you will remember that some cardiac hypertrophy with increase of blood-pressure is common to all the varieties of chronic Bright's disease, and is most marked in granular kidney, probably because this is the most chronic. But what I want to impress upon you is that this is *conservative*. The greater the blood-pressure the greater will be the amount of blood which will circulate through the kidneys in a given time, and the greater will be the output of urine. The hypertrophied left ventricle and associated vascular changes maintain a *plus* pressure in the renal vessels, and thus tend to *compensate* for the inadequacy of the organs. In our patient with such dangerous renal inadequacy this compensation is all-important; but if we place our hand over her enlarged left ventricle we find the impulse but little heaving, and the radial pulse, although persistent indicates but little increase of blood-pressure. With such poor kidneys we want a better heart. We have only a minimum of blood-pressure, and we must be most careful to do nothing that would be likely to diminish it.

If we look at the general nutrition of our patient we note that she is pale, thin, and weak; but perhaps not more so than the prolonged starvation dietary, confinement to bed, and drain of albumin would account for. An examination of her eyes, however, reveals numerous haemorrhages, and a few white spots on the retinae. These retinal changes, which are probably due in part to tissue degeneration, are important as indicating that the disease is in an advanced stage. They are always of unfavourable augury.

To sum up then our diagnosis, we may say that the prominent features of this case are: grave deficiency in the excretion of solids; a minimum of compensatory hypertrophy of the left ventricle; and marked "albuminuric retinitis."

Turning now to the *symptoms* we have been watching during the past three months—these have been what our diagnosis led us to anticipate. The grave deficiency in the urinary solids prepared us for uræmic symptoms, by which I mean symptoms due to the retention in the blood of substances which ought to be excreted

by the kidneys. Speaking generally, such symptoms do not supervene whilst the quantity of urine is normal; it is when it becomes subnormal that they are apt to occur. In our patient the daily quantity of urine has ranged from about 1100 to 1700 c.c. She has been, and is still, on the *borderland of uræmia*. The symptoms for which she was admitted—convulsions and coma—were uræmic; and during the three months she has been here, her prominent symptoms have been headache, vomiting, diarrhoea, and transient loss of vision—all uræmic symptoms.

You know that headache is extremely common in these cases of chronic Bright's disease, and it is usually most marked in the early morning. It is probably partly uræmic, due to the toxic influence of retained urinary products, and partly the result of increased blood-pressure; the latter accounting for the fact that it is so often intensified by the recumbent position. The frequently associated insomnia may be similarly explained.

Vomiting has been extremely persistent and severe. It is, as you know, a frequent concomitant of uræmia, and is sometimes associated with an inflammatory condition of the stomach; but whether this is the cause or the consequence of the symptom is doubtful. Our patient has had to be fed on milk and soda-water owing to the severity of this symptom; and at one time recourse was had to rectal feeding. Diarrhoea of several days' duration has been another symptom, also uræmic. The importance of the vomiting and diarrhoea as tending to weaken the patient, and thus to interfere with the maintenance of increased blood-pressure, must not be forgotten.

We have alluded to the more or less persistent impairment of vision due to the retinal changes, but, apart from this, our patient has on two or three occasions had that transient, almost complete amaurosis which is an occasional effect of uræmia.

In addition to these uræmic symptoms, we have had others which are mainly due to the increased blood-pressure. In most cases of granular kidney, the symptoms in the earlier if not in the later stages are, as you know, cardio-vascular—due to the increased blood-pressure which, to a greater or less extent, is an accompaniment of the disease, e.g. the palpitation, shortness of breath on exertion, frontal headaches in the early morning, and polyuria. In our patient such symptoms have

been masked since her admission by the uræmic symptoms. Still, some of them have been obtrusive. For example, on three occasions she has had severe epistaxis; this, and probably her retinal haemorrhages, being partly due to the increased blood-pressure. Then, you will notice that she has paralysis of her sixth nerve on the left side; the external rectus is paralysed, probably from a small haemorrhage near the sixth nucleus.

Lastly, with regard to treatment. In treating this patient I think we should keep three things constantly in mind: first, and by far the most important, the dangerous inadequacy of her kidneys; secondly, the small amount of compensatory hypertrophy of her left ventricle; and thirdly, that it is a case of advanced disease, and therefore alleviation is all that is possible.

Obviously the most important treatment is dietetic—to restrict her dietary and especially the nitrogenous food. For a long time she was kept almost exclusively on milk and soda-water; but she is now able to take a little chicken or fish. Such a restricted dietary is by no means always necessary in Bright's disease. In a recent acute case it is difficult to be too strict, and it is often wise to give nothing but milk and farinaceous food for long periods. Care and perseverance here is all-important, and want of it too often prevents complete recovery. In chronic incurable disease, however, although some restriction in butcher's meat and alcohol is usually desirable, a too rigid dietary, except under special circumstances, is uncalled for. In our patient, the serious deficiency in the excretion of urinary solids has made, and will, I fear, continue to make, a restricted dietary imperative.

Next in importance is rest. This by diminishing tissue-metamorphosis reduces the amount of waste products for excretion, and also tends to conserve the cardiac hypertrophy, of so much value to our patient. Warm clothing, an occasional hot bath to promote the action of the skin, and a purgative when necessary, appear just now to be all that are required, as the uræmic and other symptoms have for the present subsided.

In the treatment of the headache, vomiting, and other uræmic symptoms, which have been so persistent during the past three months, in addition to a restricted dietary, and rest in bed, we have

endeavoured to supplement the impaired renal functions by purgatives and diaphoretics. Hydragogue purgatives, remember, markedly lower the blood-pressure, and thus tend to diminish the amount of urine and the excretory function of the kidneys. In a case like this, in which so much renal inadequacy is associated with such a poor heart, their administration requires much caution. An occasional dose of Carlsbad salt or comp. jalap powder represents the most we have done in this direction ; and even now an aloes pill is used to obviate constipation rather than the more depressing morning saline.

The production of profuse diaphoresis causes much less cardiac depression, and is consequently under these circumstances a safer mode of treatment. In our patient this has always been followed by a marked amelioration of the symptoms. With profuse sweating the headaches and sickness have disappeared.

To procure the diaphoresis we used pilocarpine. The patient was stripped, wrapped closely in a hot dry blanket, covered with two more, encouraged to drink freely some hot liquid, such as weak tea ; and then pilocarpine nitrate gr. $\frac{1}{6}$ was administered hypodermically. This quickly caused a profuse sweat, lasting about three-quarters of an hour, after which the patient was dried and wrapped in a blanket. This treatment was repeated *once daily for six weeks, and during twelve days of the time twice in the twenty-four hours.* The result was eminently satisfactory, headache and sickness being so much relieved that the patient looked forward to her " pack." With the exception of the usual salivation there were no unpleasant symptomis ; and in spite of the unusually long continuance of the drug no increase in the dosage was necessary. We have used pilocarpine during the past two or three years, and consider it a safe, easy, and efficient way of producing diaphoresis. When this treatment was discontinued the patient was ordered a simple hot bath twice a week.

Although the condition of the circulation has been such as to make us most careful not to interfere with what little cardiac hypertrophy existed, at no time has there been any such marked heart failure and diminution in the quantity of urine as to call for the exhibition of digitalis or similar heart tonics. When such failure supervenes in these cases, you will remember the importance

of giving a vaso-dilator like nitro-glycerine with the digitalis.

In concluding this survey of our treatment of this case, you will note there has been no allusion to diuretics. In such advanced disease their utility is doubtful, and when vomiting is so persistent their administration is often difficult. The vascular diuretics, those which increase the blood-pressure, have, as we have seen, not been specially indicated. Digitalis was given for a few days, but as it appeared to increase the sickness, was discontinued. What appeared to be of most use was some citrate of potash in effervescent form. This relieved the sickness, and perhaps stimulated what remained of the secreting structure of the kidneys.

A CLINICAL LECTURE

ON

ADENOID VEGETATIONS IN THE NASO-PHARYNX, AND THEIR TREATMENT.

Delivered at the Victoria Hospital for Children,
June 25, 1896, by

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GENTLEMEN,—Post-nasal growths or adenoid vegetations in the naso-pharynx, and their treatment, have been chosen by me as the subject of my lecture to-day for several reasons, amongst which are the frequency of such growths, the serious complications which may be induced by their presence, their amenability to treatment, and the erroneous views entertained, and I fear sometimes taught, concerning the advisability of interference with these structures. It is a most remarkable fact that the influence for harm which these growths are prone to produce has only within very recent years been understood, for although Czermak had noticed and described growths of this nature in the naso-pharynx, it was only in 1868 that Prof.

Meyer, of Copenhagen, gave the first true description of post-nasal growths or adenoid vegetations, with a lucidity and masterly clinical descriptive power which has placed the profession under a debt of gratitude to him. Löwenberg, of Paris, should also receive the credit, which is undoubtedly his due, of aiding in drawing the attention of the surgical world to the importance of appreciating the rôle which these structures play in the causation of inconvenience and disease in those suffering from them.

"What are post-nasal growths or adenoid vegetations?" is a question frequently asked by the student. They consist in an hypertrophy of the pharyngeal, or Luschka's tonsil, which is a mass of lymphoid tissue situated on the vault and on the posterior wall of the naso-pharynx, and is constantly present in young subjects. This pharyngeal tonsil, when examined in a young child, is found to be bounded on each side by the orifice of the Eustachian tube, and to present on its surface several vertical furrows which partially subdivide it. Adenoid vegetations consist of an overgrowth or hypertrophy of this pharyngeal tonsil. This condition is most common in young children under the age of eight years. It is fairly frequent between the age of eight and the period of puberty, after which time the frequency with which the condition is met with rapidly diminishes, insomuch that the disease is somewhat infrequent between puberty and 24 years, and rare after the age of 24. I have on 3 occasions only out of 1200 cases seen marked hypertrophy of this pharyngeal tonsil in patients over 30 years of age, and never after the age of 38 years. I wish to lay special stress on the fact that though in the large majority of cases not interfered with by operative treatment, this overgrowth of the pharyngeal tonsil becomes absorbed before, at, or about the period of puberty, yet in a certain proportion, fully, I am convinced, 10 or 12 per cent., the absorption does not take place until early adult life has been reached, and in a small minority of cases not until the patient approaches the period of middle adult life. Heredity plays, without doubt, an important part in the causation of adenoids. I find that the condition can usually, when present in a child, be inferentially diagnosed to have existed in one of the parents, who will usually admit to having had a weak throat, and to having snored, and been

liable to take cold in the head during childhood. It is noticeable that the condition tends to occur among several children in the same family; thus it has been by no means an uncommon experience for me to have to operate upon two, three, or even four children in the same family for these post-nasal growths.

My experience has been that adenoids are distinctly more common in boys than in girls, in the proportion of 2 to 1, and this has been the case alike in hospital and private practice.

Post-nasal growths are encountered in two very distinct and definite types. (1) In one the growths are soft and oedematous, easily lacerable, highly vascular, prone to bleed, and they hang down as spongy papillary processes of diverse sizes and shapes, which give to the examining finger the sensation which has been aptly compared to that of touching a bunch of earthworms. This type, often figured and described as the more common, is in reality much less frequently met with than that just to be mentioned. (2) In this type the mass presents the characteristics of a distinct tumour growth; its surface is usually smooth, except where it is grooved by the vertical furrows before mentioned; it is firm, fibrous, and often slightly constricted where it is attached to the posterior pharyngeal wall. It is usually more prominent on one side than on the other, but in certain cases the tonsil is symmetrically enlarged. The mass gives the feel of a firm, tensely elastic tumour, growing from the vault and the posterior wall of the pharynx.

Microscopically, the structure of adenoids shows the closest affinity to that of the faucial tonsils, both being composed of lymphoid tissue, i.e. a delicate spongy retiform connective tissue, within the meshes of which are accumulated lymphoid cells. Two points of distinction between the histological characters of adenoids and of the faucial tonsils, are that there is a larger proportion of fibrous tissue in the latter than in the former, and also that whilst the tonsils are covered by stratified epithelium, that on the surface of adenoid vegetations is of the columnar ciliated variety.

Concerning the causes of adenoids, it is difficult to speak with any certainty. It is frequently taught that struma is the cause of adenoids *par excellence*. Whilst it is undoubtedly true that adenoids are very frequently observed in strumous

children, it is to be noted that they are as frequently met with in those who are healthy and vigorous. Again, it is stated that measles, scarlatina, diphtheria, and other specific febrile affections are prone to induce the formation of adenoids. But the probability is that though these conditions may cause increase in the size of the adenoid mass, and thus direct attention to it, that the growth was antecedent to the exanthem. We are, of course, scarcely ever in a position to state definitely at what period the growths commenced to appear; we can only fix with any degree of certainty the time when symptoms were first noticed.

This brings us to the important question: "What symptoms are commonly observed in post-nasal vegetations?" and here, at the outset, I would wish, though a strong supporter of the removal of adenoid vegetations, distinctly to disassociate myself from the number of those who see in adenoids a cause for an endless number of affections in different parts of the body, which are curable only by the removal of the vegetations. Adenoid vegetations give rise in all conscience to a sufficient number of symptoms, and in many cases to complications of such severity and fatality that there is little need to add to their number by the insertion of others, which are, to say the least, extremely doubtful.

Among the symptoms of adenoids may be mentioned defective nose respiration, breathing with open mouth, noisy respiration, with snoring during sleep, embarrassment of breathing, with semi-suffocative attacks which cause the patient to moan and be restless during sleep, and often to awake suddenly from night terrors, sluggish and delayed mental powers, obliteration of the nasolabial fold of the face, drawing downwards of the inner canthi of the eyelids, which, with open mouth, gives a dull, semi-idiotic, vacant appearance to the face, loss of resonance of the voice, and inability to correctly pronounce n, ng, m, th, p, t, k, etc., and most important of all, deafness and other aural troubles, among which may be mentioned tinnitus aurium, otorrhœa, and attacks of non-suppurative catarrh of the tympanum, causing earache. All these aural complications are the result of catarrhal conditions of the Eustachian tubes, started, or at least kept up, by the presence of the post-nasal growths always in the neighbourhood of, and at times actually pressing upon and

involving the lumen of the trumpet-shaped orifices of the Eustachian tubes. It is remarkable how frequently the deafness from which these patients so commonly suffer passes unobserved by the parents, though it is present to a marked degree. Its onset has been in some cases so gradual that it has escaped notice. It must be borne in mind that the defective hearing is the chief cause of the apparent mental dulness and incapacity of children suffering from adenoids. Several times have I been fortunate enough to have transformed the "dunce" at the bottom of the class to the bright child at or near the top, by means of the removal of a mass of adenoid vegetations. This list of symptoms of post-nasal vegetations is large and formidable. A combination of all or even several of these symptoms is diagnostic of adenoids. Still, we must honestly confess that the growths may be present and of considerable size, and yet may, in certain instances, give rise to no symptoms noticeable by the patient.

Mention may here be made of the fact that in-drawing of the membranæ tympani can almost invariably be made out in cases of post-nasal growths. The faecal tonsils, again, are very frequently, though by no means always, hypertrophied, and it is noteworthy that, however large these structures may be, it may be taken as an undoubted fact that hypertrophied faecal tonsils never by themselves enforce mouth-breathing, this being, in children, almost diagnostic of post-nasal vegetations.

On the posterior pharyngeal wall are frequently to be observed in children soft œdematosus gelatinous-looking granulations, which increase in bulk as they approach the naso-pharynx. These are, in my experience, diagnostic of the presence of considerable masses of adenoids. I have never seen them except in association with post-nasal vegetations. They are hypertrophies of the lymphoid tissue of the part, and are histologically indistinguishable from adenoids, except that they lack their ciliated epithelial covering.

There is much difference of opinion regarding the effect on the general health of the individual, as the result of the obstruction caused by the hypertrophy of the pharyngeal tonsil.

Without discussing the point I may state that I have almost invariably found that removal of adenoids improves within a very few weeks to a

remarkable extent the general nutrition of the patient, and this opinion is, I find, shared by not only medical men, but by parents and by our out-patient sisters, one of whom said to me the other day : "Operations for adenoids I used to think horrible ; but I like them now because I find that the children are always infinitely the better for the operation, even at the end of two or three weeks." This statement is in entire accord with my experience.

Methods of Diagnosis.—There are two methods in general use : Posterior rhinoscopy, which can be used probably in less than half of all cases, and practically is useless in children under six years of age and in those who are unmanageable and fractious. This method I employ less extensively than many surgeons, because I find that digital exploration, which, if quickly and skilfully done, is not really painful and alarming to the patients, is easy of performance, can be completed almost before the child is aware of what is intended by the surgeon, enables the examiner to estimate correctly the amount of the obstruction to nasal respiration, and informs him of the characters of the growth, e.g. the softness or firmness—information of the utmost value, and concerning which posterior rhinoscopy teaches us nothing.

Digital exploration is thus conducted : The patient is requested to open his mouth and to lean the head forward ; the index finger is then rapidly passed along the under surface of the soft palate up into the naso-pharynx. The operation is practically instantaneous, and is applicable to all cases, and gives information regarding which there is no element of doubt. The finger should be aseptic, for even though great care and gentleness be employed there is usually some slight staining of its tip by blood from the vascular neoplasm.

We can thus by means of the finger appreciate the size of the adenoid tumour, which may vary from a small mass the size of an almond to a bulky growth which entirely fills up the naso-pharyngeal space, and pushes downwards and forwards the soft palate. In all cases in which adenoid growths are formed it is imperative to examine by appropriate means the naso-pharynx and larynx as well as the naso-pharyngeal cavity.

Prognosis of Adenoid Growths if not Removed.

This is a point upon which it is exceedingly

difficult to speak in many cases with certainty. There can be no doubt that the prognosis depends largely upon the amount of obstruction to respiration which is produced by the mass in the naso-pharynx. If there is no marked embarrassment to nasal respiration, growths of considerable size may undoubtedly persist for years without giving rise to any untoward results. One frequently meets with very large growths in persons from thirteen to eighteen years of age, in whom they have occasioned no symptoms, nose breathing not having been obstructed. One cannot too clearly admit that a largely hypertrophied pharyngeal tonsil may never at any time produce any serious symptoms, though it persists for many years. We must allow likewise that these growths have an innate tendency to disappear at or shortly after the time of puberty, and that if no serious troubles have been occasioned by their presence before that period, there is little probability of their causing serious symptoms later in life.

Nevertheless, there is but scant justification for leaving adenoids alone. Early removal is in almost all cases the wise rule of practice. In slight cases with no symptoms it may be the part of wisdom to wait for the spontaneous disappearance of the mass. In the vast majority of cases with symptoms, delay is fraught with danger. The following are cogent reasons against delay in operating upon post-nasal growths.

Adenoid masses, large enough to give rise to obstruction to nose-breathing, have usually a most harmful influence upon the general health and nutrition, and also to some degree upon the mental development of the individual. These results persist even if the growths disappear spontaneously. They are always a serious danger to the ears and to the respiratory tract. Aural complications of various kinds, and catarrhal pharyngeal, laryngeal and pulmonary conditions are at all times liable to be induced by the presence of these growths, and the various infectious diseases—chiefly scarlatina, diphtheria, and measles—are not only more liable to occur among, but are far more dangerous to, children suffering from hypertrophied pharyngeal tonsils, than to those who are free from these neoplasms.

Treatment of post-nasal growths may be divided into palliative and operative. The former has, in my hands, yielded results of comparatively trivial

value; the latter has proved curative in almost every case, and has given me more satisfaction than almost any other surgical procedure. I will, therefore, content myself with a few remarks on palliative treatment.

In slight cases where there is but very little obstruction to nasal respiration, and in which there is reason to believe that if we can reduce the chronic inflammatory condition and thus the vascularity of the mass, we may restore normal nose-breathing, it is advisable to commence with treatment directed to this end. We may gently syringe along, or spray the inferior meatus with various dilute saline solutions at the temperature of the body, thus :

B.	Potassii Chloratis	... gr. vij
	Aquam ad	... ʒij
or B.	Sodii Bicarbonatis	... gr. v
	Sodii Biboratis	... gr. v
	Acidi Carbolici Cryst.	... gr. j
	Glycerini	... mxx
	Aquam ad	... ʒij

Or we may follow the method of Dr. Urban Pritchard and employ the following :

B.	Glycerin. Acidi Tannici	ʒij
	Aquam ad	... ʒij

One teaspoonful to a wineglass of water to be injected down the nose night and morning.

By the employment of such measures the soft, more vascular adenoids certainly in some cases seem to contract and nose-breathing improves, provided that further catarrhal attacks can be averted. This treatment is simple, and can do no harm; it may be employed in mild cases where the growths are soft and vascular. In the more common, firmer variety of adenoid tumour mass it is ineffectual, and removal of the growth is called for.

Removal of Adenoids.—This may be effected either by burning, scraping, or best, I think, by a combination of punching out, with subsequent scraping. With regard to the removal of these structures by means of the electric cautery, I can only advise this in exceptional cases, i. e. in adults who for some reason or other will not take a general anaesthetic. In such cases the soft palate and the naso-pharynx being thoroughly cocaineised, a self-retaining palate hook is introduced and the soft palate drawn forward. Then with a good

light thrown into the mouth, and by using a rhinoscopic mirror, masses of adenoid growth may be burned away with the electric cautery kept at a dull, cherry-red heat. If used at this heat there is no haemorrhage, cocaine anaesthesia suffices, and in two, or, at the most, three sittings, the greater part of the mass can be safely and efficiently removed.

It is obvious that this method demands much care and practice in order to avoid injury to important parts, and that it is applicable only in those who can be relied on to remain motionless during the performance of the operation, and in whom the naso-pharynx is of sufficient size. Still, in certain cases it has many advantages, for there is no general anaesthesia with its danger and necessarily accompanying after-trouble, and there is no haemorrhage; the operation can take place in the consulting-room, and the patient can return at once to his ordinary work without delay. The disadvantage urged against the method that the growth cannot be entirely removed is not a serious one, as complete removal is not really as necessary in adults as it is in children, in whom there is so great a tendency to recurrence of the growth should any particles be left behind.

In the vast majority of cases removal under general anaesthesia is the best method of treatment, and in considering the subject of removal of adenoid growths under general anaesthesia we may discuss—(1) The position in which the patient is to be placed. (2) The choice of an anaesthetic, with the degree of anaesthesia required. (3) The instruments required in the performance of the operation. (4) The after treatment, and (5) The results of the operation.

It may be stated at once that it is advisable, and, in cases where already at the time of the operation any aural complication exists, usually imperative, to remove, as far as possible, every portion of the post-nasal growth. The younger the patient the more necessary it is to follow out this rule of practice. When hypertrophy of the faecal tonsils co-exists it is usually advisable in younger and weakly patients to amputate these structures without an anaesthetic a few days before proceeding to deal with the pharyngeal tonsillar hypertrophy. In more vigorous and older patients for whom the shock and loss of blood of the two operations is not feared, it is often advisable to

first amputate the tonsils and then remove the adenoids under one administration of the anaesthetic.

1. The posture of the patient while under general anaesthesia. This is a point of immense importance, for we cannot disguise the fact that whilst the operation without anaesthesia is quite devoid of risk, anaesthesia introduces an element of danger. On the Continent the operation is usually performed without the administration of a general anaesthetic. In this country, however, this is seldom done, as it is not only difficult, if not impossible, to thoroughly remove every particle of growth without general anaesthesia, but there is risk of great injury to the child owing to the mental shock occasioned by such a procedure.

Having seen chorea develop twice, and having been informed by the parents that in other patients a condition of hysterical crying and mental irritability had been noticed immediately after the operation, which condition lasted for months, I have abandoned operating without anaesthetics. The risk of a general anaesthetic is that of blood gaining entrance into the lower respiratory tract, and thus causing asphyxia at the time of the operation or inflammation of the lungs later on.

Three aids in warding off this danger are an experienced anaesthetist, skilful and rapid sponging, and a safe position to be maintained throughout the operation.

The safest position, in my opinion, and the most convenient for the operator who uses the Löwenberg forceps or the curette, is undoubtedly that in which the patient lies on his back with the head hanging over the end of the table. This position of the head somewhat increases the bleeding, but it absolutely prevents blood entering the larynx and bronchi, as to do this it would have to run up-hill. In this position the blood runs out of the nose or mouth, carrying with it fragments of the neoplasm. The bleeding is often furious for a moment, but ceases almost invariably soon after the head is raised. The position recommended by Sir W. Dalby and Mr. Braine, in which the body is flexed on the thighs and the head bent forward so that the blood escapes from the mouth into a basin placed between the knees is also a safe one when gas is employed, but is only suitable for mild cases in which the natural

or artificial finger-nail is employed, and is, I think, inferior for other reasons to the dorsal position with dependent head. Any other position than the two named above I look upon as unsafe, and I strongly recommend the first described.

2. The choice of an anaesthetic, with the degree of anaesthesia required. We will consider chloroform, ether, gas, and gas and oxygen. Chloroform is the most handy and the most easily administered. It is not to be pressed to full surgical anaesthesia, thus the swallowing and coughing reflex is not to be totally suspended. There have, however, been even in careful hands so many deaths from this anaesthetic during the removal of adenoids that we must admit that its administration is by no means without a certain element of risk. I have used it hundreds of times, but always with "fear and trembling," which by the way is not an unwise precaution when dealing with this powerful and dangerous drug.

Ether, I confess, I dislike intensely for adenoids, for these vascular structures under the irritation caused by its vapour become distinctly more vascular and swollen, and a large amount of frothy mucus is poured out, which increases the frequency of the necessary sponging.

There is in my mind no doubt that the haemorrhage with ether is far greater than with any other anaesthetic, and when to this we add the abundance of the frothy mucus, and the irritating effect of the ether vapour on the already congested mucosa of the upper respiratory passages, I think we may conclude that ether is by no means so suitable an anaesthetic for this operation as it undoubtedly is for many surgical procedures. Gas administered as a preliminary to ether to a certain extent does away with some of my objections to this anaesthetic.

Nitrous oxide has proved in my experience the safest and best anaesthetic for general use. It can be given to all patients, and has no unpleasant after effects. In fact, it is the rule in my clinic, both at this hospital and at Charing Cross Hospital, for the patients to walk home within a few minutes after the removal of adenoids under nitrous oxide. The objection to nitrous oxide, that it does not allow the operator sufficient time for the complete removal of the growths, is, I think, now much weakened by the introduction of the combined administration of gas and oxygen. Still, I find that provided the gag be introduced before the

administration of the gas is commenced, sufficient time is allowed to a fairly rapid operator if the adenoid tumour be not too large and firm.

Gas and oxygen together form, in my opinion, the best anaesthetic for most cases of adenoids. I have employed the method in more than one hundred cases, and am much pleased with it; I have found it accompanied by less haemorrhage than gas or ether, that it gives ample time for the removal of the growths, has no unpleasant after effects, and is absolutely safe. My friend Mr. H. Bellamy Gardner,* who has given it in very numerous cases for me, has kindly come down this afternoon to administer the anaesthetic to eight children of various ages upon whom I will operate before you at the conclusion of this lecture, and you will be able to judge for yourselves of the value of the anaesthetic.

3. With regard to the instruments employed, I will mention four only: (1) the natural finger-nail; (2) the artificial finger-nail invented by Sir W. Dalby; (3) the forceps (Löwenberg's); and (4) the curette.

The natural finger-nail I find of use only in clearing away the last vestiges of the growth after the greater part has been removed by some more powerful means. Dr. Pritchard recommends hardening the nail in alcohol, but in spite of this I have so frequently bent back or broken my right index finger-nail that I have abandoned the use of this simple method except in so far as I have stated above. The artificial finger-nail of Dalby which I show you is employed by several surgeons. It is very efficient, but I rarely make use of it, as with it placed upon my finger tip I feel that I have to a great extent lost my normal appreciation of the sense of touch. It is, however, in unpractised hands the safest of all instruments, as with it injury to other parts can scarcely be inflicted. It may therefore be recommended to beginners.

The two instruments which I now exclusively use are Gottstein's curette and Löwenberg's forceps.

Gottstein's curette is this fenestrated instrument, triangular in shape with the cutting edge on the inner margin of the base of the triangle; it is the most generally useful of all instruments for the removal of adenoids. The flat triangular part of

this instrument guided by the forefinger is introduced into the naso-pharynx from the mouth, and being well pressed upwards against the vault of the naso-pharynx is firmly drawn from before backwards so as to thoroughly scrape away the whole adenoid mass, which falls into the fenestra of the instrument. After two or three motions in the antero-posterior direction, sponging requires to be carried out to avoid the accumulation of blood in the part. In the majority of cases the finger is introduced to scrape away any fragments that may remain.

For soft growths Gottstein's or some similar curette, of which there are many, amply suffices, but when we encounter a firm adenoid tumour I find it always advisable and frequently indispensable to employ some form of cutting forceps. Of these I think Löwenberg's, in spite of the many modifications of it which have been introduced, remains the best. The forefinger of the left hand being introduced into the naso-pharynx as a guide, the forceps are passed above and behind the soft palate from the mouth; they are then widely opened and pressed up into the adenoid tumour, out of which they are made to bite a considerable piece. This manœuvre is repeated say four or five times, when the curette is used to scrape away the rest of the growth; or, if preferred, the whole mass may be removed piecemeal by the forceps, though this is scarcely possible under gas and oxygen anaesthesia, owing to the longer time required. To sum up, for soft growths Gottstein's curette is sufficient; for firm adenoid tumours it is well to punch several pieces out of the mass before employing the curette. A great advantage in using the forceps is that as there is required a certain combination of tearing, punching out and twisting, the haemorrhage is less than with the curette, which cuts more like a knife.

When I fear haemorrhage in a weakly child I employ the forceps for the whole operation.

4. The after-treatment. The patient must be watched for some time after apparent recovery from the anaesthetic, especially when chloroform or ether has been employed. Some blood is usually swallowed after the operation is completed. This, if in considerable quantity, causes vomiting some hours afterwards. For the first twenty-four hours the patient should remain in bed, and only cold fluids or semi-solids, milk, custards, bread and milk, &c., should be given. For three or four

* Mr. Gardner's contribution on the subject will appear in our next issue.

days confinement to the house is advisable. Owing to the blocking of the nasal passages with blood-clot, fragments of growth, and mucus, nasal respiration is often rendered for a day or two even more difficult than before the operation. At the expiration of twenty-four hours, I advise that the nose be sprayed thrice daily with the following, used at blood heat.

B. Sodii Bicarbonatis ...	gr. v
Sodii Biboratis ...	gr. v
Acidi Carbolic Cryst. ...	gr. j
Glycerini ...	ijxx
Aquam ad ...	3j

At the end of three or four days nose-breathing usually becomes restored. Shortly after the operation, in about 2 per cent. of cases, pain in the ear is complained of. Politzerisation usually relieves this at once, though sometimes middle-ear suppuration results, due probably to the entrance of blood into the tympanum through the Eustachian tube during the performance of the operation. Aural complications I have found quite rare, I think one cause being that I never employ that baneful apparatus, the nasal douche, which is, I am sure, a cause of much middle-ear trouble. I think even the use of a syringe inadvisable. Deafness due to Eustachian obstruction generally yields within three to ten days after the operation. Should it not do so at the expiration of this time, but not earlier, Politzerisation or the Eustachian catheter may be employed.

5. The results of the operation are in fully 99 per cent. of all cases eminently satisfactory. Nose-breathing becomes re-established, all symptoms of obstruction cease, the liability to aural complications disappears, and the general health rapidly improves. If mouth-breathing persists it may generally be cured by the diligent use of the spray above mentioned, or by cauterisation of the inferior turbinals. Out of over 1200 cases I have fortunately had no fatality, thanks to the skill and care of my anæsthetists. I have three times only had momentary trouble with the anæsthetic (in all three cases chloroform was used). In nearly 1 per cent. of the cases the temperature has risen considerably the night and morning after the operation, the glands of the neck have swollen and become painful, but I am glad to say that these unpleasant symptoms have disappeared without causing any real trouble. I cannot recall one

single instance in which three weeks after the operation the parents regretted its performance. In its almost invariably good results I know of no operation in surgery which surpasses, and of few which equal it, and fully convinced of its beneficial influence on the general health, of its power of warding off aural complications with their dangerous sequelæ, and of its almost infinitesimal risk in practised hands, I heartily commend the operation to your favorable notice and urge on you the necessity of its performance in all cases of post-nasal vegetations with symptoms of obstruction or of aural complications.

REVIEW.

THE "LANCET" AND THE HYDERABAD COMMISSIONS ON CHLOROFORM.

Being the Report of the "Lancet" Commission appointed to investigate the subject of the administration of Chloroform and other Anæsthetics from a Clinical standpoint, together with the reports of the First and Second Hyderabad Chloroform Commissions.

(Concluded from p. 271.)

THE clinical side of the investigation was undertaken, we are told, to supplement the experimental portion of the Report, and further, to range into line the clinical results with the experimental. An effort was made to arrive at statistics dealing not only with the fatalities occurring under or during the use of chloroform and other anæsthetics, but also with the cases of normal anæsthesia, and those which revealed some departure from the normal, but yet did not give rise to death or danger. It was thought that three sources of information were open to the Commission: (1) published cases, (2) notes made by medical practitioners, and (3) notes kept at the hospitals. The means adopted to obtain the information from these were: careful collocation of published cases; circularization of the profession; investigation of the hospital registers. The published cases were examined and reduced to a synopsis of facts, the published records being used to mutually check and expand the others. These synoptical tables include the elaborate report published by Snow,* which with the

* On Anæsthetics, page 123.

report issued by the Royal Medical and Chirurgical Society† in their "Transactions" form one series; then the reports from the current literature are classed together in the second series; while the hospital cases, and cases reported from private practice, are grouped in separate classes. The Report before us brings out the regrettable fact that few of the larger hospitals, and hardly any of the smaller ones, have made an effort to keep a continuous record of their cases of anaesthetization; a still more astonishing fact is, that no particular notice appears to be taken even of the fatalities; the usual stereotyped coroner's finding that "no blame is attached to the medical officer representing the hospital" appears to be considered as absolving him from even studying the case with a view to lessening the future mortality under anaesthetics! Taking the cases as a whole, we find statistics dealing with (*a*) death due directly to the anaesthetic, (*b*) death due indirectly to the anaesthetic. "Untoward" cases, again, are classed as directly or indirectly due to the anaesthetic. These cases are considered in Part I, which deals with chloroform, and an attempt is made to ascertain what are the determining factors giving rise to death or danger. Age and sex are first considered. It is pointed out that although more men actually die from anaesthetics than women, this may be accounted for by the fact that they are more exposed to accident, and more liable to undergo surgical operations; although it seems hardly likely that the proportion of three deaths in men to one in women can be entirely explained by this. Most men are said to die under chloroform between twenty-five and fifty, most women between twenty-one and forty-five. Under the heading of geographical distribution, a long list of places from which deaths are reported is given, but no definite conclusion is arrived at as to possible telluric or climatic influences at work in causing the deaths, and the Commission appears to regard any conclusion drawn from their list as not wholly reliable, since returns have not been universally made. No doubt the places from which the returns come are those where most activity exists, and therefore most surgical work is being done; hence it would be an obvious fallacy to believe that they were the most prone to fatalities.

The methods employed for giving chloroform are

dealt with on page 91. They include such modes as chloroform poured on a handkerchief, towel, &c. &c., and we note that the recorded deaths from chloroform being administered without an inhaler are rather more than double those when an inhaler is used. With such evidently faulty methods it is surprising how low the rate of mortality is shown to be. Indeed, the perusal of the cases published in the synoptical tables of this Report impress one with the fact which we do not find stated in the Report, that a very large number of the deaths must have been due to faulty administration, if not to carelessness. We may hope that these facts having been brought to light by the Report will, at least, impress upon the profession the danger of slipshod methods and the ever-increasing responsibility of the medical man who undertakes to give chloroform while surgical operations are in progress. We are told in the Report that no very distinct proof is present that the open method is more dangerous than that which involves the employment of some regulating inhaler, because it is alleged that the use of the open method is so much more common than that of an inhaler. This may be so; but the fact remains that of the cases of death reported in the tables, a very large proportion were clearly those in which a faulty method of administration was adopted.

A very important part of the Report is that which deals with the causes assigned for deaths under chloroform. While no very definite predisposing causes are shown to exist, very distinct evidence is brought forward to demonstrate the fact that heart failure has been very commonly observed to occur, either simultaneously with cessation of respiration or before the rhythm of breathing was affected. It has been alleged by those who hold that respiratory failure is the only way in which chloroform kills, that no instance of primary cardiac failure under chloroform has been published. This Report furnishes a number of such cases. To us it appears in the last degree improbable that all the observers who distinctly state the heart failed before the respiration ceased, should have been afflicted with an epidemic of being unable to notice the sequence of events. Thus 227 cases of cardiac failure before respiratory failure are mentioned, to 80 of primary respiratory failure, and to 77 cases of simultaneous cessation of circulation and respiration. But

† *'Med.-Chir. Transactions,' vol. xlvii, p. 377.*

before we leave this portion of the Report we must say that a perusal of the synopsis of cases certainly shows that many instances reported as primary cardiac syncope appear to be examples of incomplete anaesthesia, or anaesthesia becoming incomplete through the patient partially resuming consciousness. No doubt many of these so-called primary cardiac failures are really instances of reflex inhibition of the heart, due in some cases to surgical shock, in others to faulty position, and in others to the onset of vomiting. In the valuable notes and conclusions appended to the synoptical tables, these considerations are fully gone into, and a painstaking effort made to do justice to both sides of the controversy, and to arrive at not only the apparent, but the actual cause of death.

No fresh information can be said to be given by the tables dealing with the methods of resuscitation employed in fatal cases; where all failed it is naturally impossible to say which was better than another, but the subject is again taken up in the section dealing with the "untoward" cases. Here unquestionably the employment of artificial respiration and the method of partial or entire inversion proved most successful. We notice various cases in which brandy or ether was injected hypodermically when the patient was apparently suffering from an excessive dose of the anaesthetic, clearly a faulty method to add further poison to a pre-existing toxæmia. It has struck us that in some cases unwise efforts at resuscitation have been responsible for the fatal event, and certainly some cases reported appear to bear out this suspicion.

The consideration of the nature of the operation is an important part of the Report. An appalling number of deaths are recorded while the most trivial operations were in progress, notably the extraction of teeth, reduction of dislocations, amputation of fingers, dressing of wounds, &c. Unless we believe in the old idea of idiosyncrasy, it is painful to find human lives sacrificed to an anaesthetic wholly out of proportion to the gravity of the operation for which it is given. One of the conclusions at which Dr. Dudley Buxton arrives, is that nitrous oxide should be employed for minor surgery, and should replace chloroform in dental surgery. The perusal of this ghastly list of persons done to death by chloroform while their teeth were being removed, should more

than justify such a conclusion. Possibly owing to the fact that the nature of nitrous oxide has been for many years imperfectly understood, one comes across persons who express no fear of chloroform, but an intense dread of the lesser and safer anaesthetic, nitrous oxide. Reports unhappily do not convince the ignorant; but we hope that this Report will, in the case of the profession, go some way to rid them from the habit of giving chloroform in cases where nitrous oxide could and should be employed.

The existence of death from surgical shock, which was denied in the Experimental part of the Report, appears to be fully proved in the Clinical part, and some very interesting reading is afforded dealing with this matter. In many instances the patient appears to have died from insufficient chloroform having been given, the conveyance of impressions along sensory nerves having taken place, with the result that respiration and circulation were simultaneously inhibited.

While on the subject of operations a very important matter is touched upon in the Report, that is, the responsibility of the person giving the anaesthetic. Quite a number of instances are given where obviously the person who gave the chloroform was quite incompetent for his office, the officiating surgeon accepting the responsibility, which he should not have conscientiously assumed. Although undue fuss and anything approaching terror or timidity must, of course, be avoided, it seems most undesirable that the person who gives the chloroform should have the hardihood which induces "fools to rush in where angels fear to tread."

The lists of post-mortem examinations are of little value, at least from the point of view of advancing our knowledge, for in all cases they are made too long after death to reveal lesions peculiar to the mode of death produced by anaesthetics. One might almost imagine the average necropsies in these cases were made rather for the purpose of exonerating the medical men present at the time of the operation than to increase the sum of our knowledge concerning the pathology of death under chloroform.

Perhaps one of the most interesting features of the report is the grouping of deaths under chloroform in such a way as to show those which occurred in the first degree of chloroform narcosis, in the second, in the third, in the fourth, and in the fifth

In the first stage we are told the form of death is that of a sudden failure of circulation and respiration ; it is spoken of as the "shot down" form of death, and is compared to the effect of gunshot wounds in the brain, thus recalling some interesting work done by Professor Horsley, and communicated to the Royal Medical and Chirurgical Society in July, 1895. The mechanism of such fatalities, the Report would have us to believe, is that a sudden intake of chloroform overpowers the medullary centres, not having time to be eliminated. This theory, although reasonable, has not, we think, been proved ; certainly many fatalities reported in this stage are deaths which may be put down to fear. Upon this subject an important point is advanced in the Report, and that is the somewhat curious fact that although deaths ostensibly due to fear occurred to patients under chloroform, such accidents are not noted in corresponding cases of persons under ether. Certainly it is very exceptional to find these fatalities under ether, and one would expect instances would have shown themselves in the many thousands of cases recorded by Julliard and the reports collected by Gurlt from the German hospital statistics. If we may then assume that they do not occur under ether, the theory of deaths from fear in the first stage of chloroform narcosis will have to be reconsidered, and the cause imputed rather to the anaesthetic than to the fright.

The stage of struggling, or the second degree of narcosis, is accredited with killing many patients from general muscular spasm, imposing impediment to respiration and interfering with the due performance of circulation.

Death in the third or surgical stage of anaesthesia is referred to failure of circulation, or failure of respiration, and the Report definitely states that primary heart failure has in the majority of cases been noticed.

The fact that deaths are reported as having occurred in the fourth degree of chloroform narcosis certainly points to very careless administration of the anaesthetic. Surely under no circumstances is an anaesthetist justified in allowing his patient to pass into this stage. One occasionally hears the injunction to "push" the anaesthetic, because the patient is not relaxed. When this is done the dangerous zone is treacherous upon, and the patient's life jeopardized. Any amount of relaxation can be

obtained in the third degree, if the administrator and the surgeon will have the patience to wait for the chloroform to take its full effect, and for the establishment of a proper balance between elimination and supply. The sudden deaths occurring without warning in the later stages of anaesthesia, are certainly in some cases due to a sudden pushing of the anaesthetic, thereby increasing the intake before the patient is able to throw off sufficiently quickly the chloroform already in the tissues.

In the fifth degree the death is due to failure of respiration, and is, according to the report, "gradual," and due to overdose, or to interference with the elimination of the drug. This question of elimination we are glad to see ventilated in this Report, for most of the works dealing with the subject of chloroform, while they impress the importance of never allowing an excessive dose to be administered, fail to point out the possible dangers of overdose by accumulation, in other words by failure of elimination. Certainly, as we are told in the Clinical Report, persons suffering from emphysema and from renal inadequacy are prone to this form of "storage" toxæmia. The emphasis given in the preliminary or experimental part of the volume to the importance of watching respiration, applies to these cases of "storage" toxæmia, for gradual respiratory failure must take place, and if observed soon enough, can be successfully dealt with. Unfortunately, the association of a dilated and feebly acting heart with pulmonary dilatation, renders the patient more likely to succumb to this mode of poisoning.

Deaths during dental operations and during the attempted reduction of dislocations are very liable to take place in the second degree of narcosis. Upon this subject it is said the mechanism of such fatalities is probably of the nature of a reflex, and arises from peripheral stimuli being conveyed along nerve channels, which, had the patient been completely under the influence of the anaesthetic, would have been closed, as far as the production of reflex inhibitions are concerned.

A certain number of deaths are said to arise from respiratory spasm, initiated by a too concentrated vapour, or by the presence of products of decomposition in the chloroform employed.

Speaking of the vomiting which in some cases occurs just before death, the Report says, "The

occurrence of vomiting even when it does not lead to the sucking in of the vomit into the air passages, seems in many cases to have coincided with the instant of death. It may of course be a phenomenon of dissolution, just as ejection of the faeces and urination often are; but, on the other hand, it is to be remarked that when vomiting comes on in cases where no fatal result occurs, there is a marked deterioration of the pulse and fall of blood-pressure."

The last mode of death cited is that following the addition of fresh chloroform to the lint or inhaler in use. "Many cases are recorded, when during the operation the patient showed signs of returning consciousness, in some instances even sitting up, and the anæsthetist at once re-applied the chloroform. The death which ensued is no doubt due either to overdose or reflex shock."

Referring to self-administration of chloroform—a habit unfortunately only too common amongst medical men—the Report justly says, "No words can be too strong in speaking in reprobation of this terrible habit."

At the time of the first cablegram from India, dispatched by Dr. Lauder Brunton, in which he announced that no heart failure occurred under chloroform, a deduction from the experiments he had witnessed, a lay paper remarked, "To take chloroform has now been shown to be as safe a proceeding as to take a tumbler of whisky and water." Upon this we cannot refrain from quoting from the Report before us, "Among the causes of death, we would say of *preventable* death, shown only too plainly in the records, is the employment of chloroform by persons either quite unfamiliar with its dangers, and the necessary precautions to be adopted to avoid these, or only partly versed in its use. Many cases of dentists, who held no medical qualifications, and of persons wholly untrained in medical and surgical work, are recorded as having given the chloroform, and therefore been largely or wholly responsible for the fatal result."

The theory, still dear to the lay mind, that a person who has taken chloroform with impunity once is free from all subsequent risk, is negatived by the evidence of the Report, which records deaths of persons who have repeatedly taken chloroform,

in one instance of death occurring at the fifth administration.

Pages 104 to 156 give the concise record of 733 fatalities associated with chloroform—an unique tabulation, and one of the greatest value. Perhaps one of the greatest merits of the Report is that it aims at giving facts, and at grouping them in such a way as to suggest evidence rather than to force conclusions. Of the series of synoptical cases it may be truly said that "he who runs may read," and it may not be too sanguine a hope to express that the profession to whom are entrusted the lives of thousands of persons who have to be anæsthetised year by year will take the trouble to study the cases, and come to some clear conclusion as to what anæsthetics are the safest, and what methods of giving them are most free from danger.

It would be tedious to go in detail into the list of "untoward" cases, although we agree with the Commissioner who writes that they form "probably the most important part of our report."

Part II deals with Ether. It is framed upon the same lines as Part I, but happily a very much smaller rate of mortality allows its compression into some ten pages. A point of considerable interest is that the deaths under ether do not appear to have arisen from causes which are usually believed to tend to a fatal result when that anæsthetic is used. A large number of fatalities arose in the cases of persons so exhausted from accident or disease that they were given ether because it was felt that they could not stand chloroform. Of these some appear to have been moribund at the time the etherisation was commenced. We find no record of that undue struggling which, according to the compilers of the experimental part of the volume, would appear to prohibit the employment of ether in surgery. Few, if any, sudden deaths are recorded under ether, but some few cases of incomplete anæsthesia are given with a fatal result. It is noteworthy that the Report does not contain evidence of any large proportion of deaths from pulmonary affections initiated by ether. Upon circulatory failure we are told "a comparatively small number of cases are reported in which ether anæsthesia is reported to have caused circulatory failure." Upon ether and renal disease the Report says with regard to the kidneys, a granular condition was reported in two cases,

cystic disease in one ; in this the vessels at the base of the brain were affected. "In no case are reliable facts given pointing to nephritic trouble following the use of ether."

Before dismissing chloroform and ether, we may refer to the concluding paragraphs of the Report, which give tables dealing with the relative number of deaths under various anaesthetics ; from these it will be seen that chloroform is still used far more frequently than ether, but the death rate of the former is to the latter disproportionately large, even when allowance is made for the fact that chloroform is the most commonly used of all anaesthetics.

The statistics of administration without fatal or "untoward" symptoms are of great value as supplying a basis of comparison between the different anaesthetics, and we only regret that the Report does not furnish fuller tables. If the large hospitals in the metropolis and manufacturing centres were to keep sufficient statistics, we should soon be able to furnish returns of the greatest value, comparable to those published year by year by Gurlt, in Germany.

One remark may be made with regard to the nitrous oxide fatal cases, and that is that most of them do not appear to have resulted from nitrous oxide at all. The less commonly used anaesthetics and mixtures are dealt with in the Report, but space prevents us dealing with them in detail.

We will bring the present review to a close with some quotations which deal with such important matters that we should fail in our duty were we not to place them before our readers. "If, on the other hand (as sometimes but rarely happened), there was sudden failure of the heart's action, the breathing still continuing, death supervened practically without warning. This mode of death, which was exceptional in the experiments on animals, is the most frequent in the human subject." So said the report of the Royal Medical and Chirurgical Society, and upon it the present Report says, "The experience of the Hyderabad Commission gave no evidence of this form of death in experiments on the lower animals, and Surgeon-Lieutenant-Colonel Lawrie had not in his Indian practice met with an instance among human beings. In the cases we have reported it would appear that such deaths do really occur in temperate climates, and with frequency." "The evidence in favour of heart

failure occurring under ether seems to be unsatisfactory, for when syncope is said to have occurred, it appears that the violence of the struggling was sufficient to have led mechanically to heart failure." "As to death occurring during partial anaesthesia from surgical shock, we submit that there is much evidence in the cases we have cited above to prove that such an accident is frequent, and occurs alike under chloroform, under ether, and under nitrous oxide." "In the larger number of fatalities the operation was of a trifling nature, and this fact suggests the possibility in some of this number of less care, and precaution, and individual attention to the anaesthetic than are associated with the graver operative procedures." "It has long been held and taught that parturient women and young children were almost exempt from the perils of chloroform. But the reports before us and those supplied by the tables derived from medical men in private practice appear to negative this supposed immunity."

This valuable monograph, for which we are indebted to the generosity of H.H. the Nizam of Hyderabad, the energy of Surgeon Lieut.-Col. Lawrie, the enterprise of the "Lancet," and the unstinted labour of the distinguished men who have so successfully accomplished a work of such great difficulty, concludes with the following six deductions, which Dr. Dudley Buxton, the Commissioner for the Clinical part of the volume, thinks may be drawn from the Report :—

1. That the death-rate under anaesthetics heretofore has been unduly high, and may, by improved methods and greater care, be lowered.
2. That ether when properly given from an inhaler, permitting graduation of the strength of the vapour, is the safest anaesthetic in temperate climates for general surgery.
3. That nitrous oxide gas should be employed for minor surgery, and should replace chloroform in dental surgery.
4. That chloroform, when given by a carefully trained person, is a comparatively safe body, but is not in any case wholly devoid of risk.
5. That no age or nation is free from danger under anaesthetics.
6. That the perils of anaesthetics, however slight, demand that the undivided attention of a duly qualified and trained medical man should be given to the administration of the anaesthetic.

THE CLINICAL JOURNAL.

WEDNESDAY, SEPTEMBER 2, 1896.

A CLINICAL LECTURE ON CANCER OF THE TONGUE.

Delivered at St. Thomas's Hospital by

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GENTLEMEN,—Carcinoma of the tongue, in contrast to sarcoma, is a common affection, and, unfortunately, in this situation is of comparatively frequent occurrence. This is possibly in part dependent upon the fact that the tongue is a very vascular structure, and is maintained at a high temperature. Whatever the cause may be, the disease is apt to develop rapidly, and to determine the death of the individual at no very distant period afterwards. It is a curious fact, for which there is no adequate explanation, that the squamous-celled variety of carcinoma commonly called epithelioma, is invariably met with in the tongue. It is met with and may occur in any part of the organ, in the floor of the mouth, or in the half arches of the palate. There are, however, certain places in which the disease is more commonly met with than in others; for instance, the border of the tongue is involved about four times more frequently than is any other part of it. It somewhat rarely commences in the root, and the anterior portion of the tongue is not a frequent site, probably because the posterior part of the lateral border comes more intimately into relation with the teeth, the anterior part lying somewhat beneath their level. The disease is much more common in men than in women; perhaps 80 to 90 per cent. of the subjects of cancer of the tongue are men. The common idea that smoking is a frequent cause of cancer is not sufficiently verified to account for this difference in sex. In countries where women commonly smoke, many of them as much as the men, there is the same difference between the sexes in the

matter of cancer of the tongue. The disease is not met with in young persons. A few instances have been recorded in which persons of 25 to 30, generally nearer 30, have suffered from true carcinoma, but it is so rare at that period of life that it may be neglected as a clinical fact. The most frequent age for carcinomatous growths in the tongue is between 45 and 55.

It is liable to be caused by any long-standing continuous irritation, and, as you would naturally suppose, it is more likely to occur in a tongue which is antecedently unhealthy. In this connection it has been very well said that both syphilis and excessive smoking may produce conditions likely to predispose to the formation of cancer.

I have referred to the condition known as leukoplakia, or what is called smoker's tongue, in another lecture. There is no reliable statistical information as to smoking otherwise producing cancer except in so far as it gives rise to this diseased condition, which may be considered a pre-cancerous state. Among the contributory causes of superficial glossitis are: ulceration of the tongue, caries or tartar of the teeth, the irritation of the rough end of a clay pipe, excessive spirit-drinking, or the application of caustics; the simple ulceration brought about by any of these causes may drift into carcinoma. Of all the antecedent or pre-cancerous states, perhaps that known as leukoplakia is the most common, and if the prior state of the tongue were looked into, in all cases probably this one would be found to exist in one-fourth of all the cases of cancer of the organ. Some area of this leukoplakic condition ulcerates, and the invasion of epitheliomatous elements presently stretches into the mucous, submucous, and muscular tissues, transforming the simple into a malignant ulcer.

It is not of very much importance how the disease actually commences. I have not had an opportunity of noticing it at a very early stage, but it may begin as an abrasion, a crack, or a fissure which refuses to heal under any kind of treatment; or (much more rarely) a small, hard nodule or mass may be felt in the tongue. A frequent condi-

tion, also, in which cancer makes its appearance, is in a patch of superficial glossitis which ulcerates and refuses to heal, a slight amount of induration takes place, increasing in amount in the base and margins of the ulcer, and after an interval the more distinctive features of a cancerous sore will appear. In the same way a papilloma, which for a long time may remain perfectly innocent, will become transformed into epithelioma. So long as the epithelial elements are directed towards the surface no harm ensues, but at some time which cannot be exactly observed, and for reasons we cannot give, the epithelial cells begin to invade the deeper layers of the mucous membrane, then the fibrous layer, and then the muscular layers by degrees, and a malignant tumour is the result. The more deeply this invasion extends, the more serious is the disease, and the more extensive is the glandular implication. The glands are implicated earlier when the disease is in certain parts than in others; perhaps the glands are affected earliest when the cancer is in the floor of the mouth, and least early when the dorsum is affected. It is noteworthy to remember that in this case the lymphatic glands on both sides will probably be involved, since the lymphatic plexus at the base of the tongue drains into the glands of both sides of the neck, an important practical point in the treatment of cancer at the back of the tongue. The lymphatic glands become affected in cancer of the tongue earlier than when any other part of the body is the seat of the malady; and I think we may safely say that two or three months after the disease is first noticed the lymphatic glands which drain the invaded area will be also implicated. There is no doubt that in some parts of the tongue, the implication of the lymphatic glands may be delayed for six or eight months, but such cases are comparatively rare.

The diagnosis of the disease may at first be exceedingly difficult, and it is in the early stage that we most desire to recognise it. When in an advanced stage, the appearance of the ulcer is very characteristic. The base and margins are defined by a very marked hardness, very different from, because more accentuated than, any inflammatory induration. The surface of the sore presents different characters according to the activity of the disease. In a comparatively quiescent condition it will show a warty surface, with perhaps a few imperfect granulations here and there,

while in the depth of the ulcer there is often a sloughy appearance, due to necrosis of the masses of epithelial cells which have invaded the tissue. The discharge is not usually abundant, but is generally exceedingly offensive, as are all discharges coming from the breaking down of epithelium. The pain is generally of a severe character, though not always so at first, and there is presently difficulty in speaking and eating, especially when the tongue has been considerably invaded. When the disease begins on the under surface, or in the floor of the mouth, its mobility will be much diminished, and the functions of the organ correspondingly interfered with. Very severe pain is felt not only in the tongue itself, but it is also referred by the auriculo-temporal nerve to the ear and to the side of the head. The irritation present in the mouth causes an abundant discharge of saliva, especially in the later stages of the disease, which proves a distressing complication. The mouth is filled with foul-smelling secretion, a portion of which dribbles away; the rest is swallowed, and completely disorganises the gastric functions. The inhaling of the foetid atmosphere in the mouth often produces irritation in the respiratory apparatus, and may proceed so far as to cause septic pneumonia.

With regard to the duration of life in these cases, I have told you it is short. If no interference be allowed, and the disease progresses unhindered to its termination, it will end the individual's life in the course of a year or eighteen months. By far the most frequent manner of death is simple exhaustion, produced by the profuse discharge of saliva, the inability of the individual to consume sufficient food, the absence of an adequate amount of sleep, consequent on the continuous pain which the individual suffers, and also in frequent instances by the occurrence of haemorrhage, not commonly very severe, but repeated attacks materially increase the exhaustion. Sometimes, but not frequently, death takes place from a sudden rush of blood, caused by the invasion of the tonsillar artery or the internal carotid. I recently had a poor fellow under my care who lost his life in that way—a comparatively young man, who had been treated for a long time under the idea that he had syphilitic disease of the tongue, and when he came to me it was too late to perform any operation. He was a man who

had everything that great wealth could procure, and he died of haemorrhage in the course of a few seconds.

Complete removal in these cases certainly prolongs life. Taking the average of cases, life is prolonged for at least six months. Of course there are many in which, the conditions being favourable, the patient does not die, and recurrence does not take place for a much longer period, perhaps two or more years intervening. Unfortunately, however, as a rule recurrence does take place, either in the mouth or the lymphatic glands. Local recurrence is much the less frequent, but it is not so infrequent as is generally supposed. Kocher estimates recurrence takes place in 60 per cent. of the cases, while Winniwarter puts it at 90 per cent. I think the last is the more correct, because the more favourable statistics are based on the assumption that no recurrence within a year means a cure; but even under those circumstances only about 10 per cent. can be found cured, even in this modified degree. There are a few cases in which recurrence does not take place at all; and I can recall two instances in my own experience, both of them men of 60 years of age, from whom I removed half the tongue. In neither was there any evidence of enlargement of lymphatic glands, so that the disease was taken in a comparatively early condition. On microscopical examination the growth proved to be carcinoma, and the clinical characters were clearly those of that malady. In one of these instances the gentleman lived for five years, and then died of apoplexy, having had no return of cancer during his life. The other patient died between six and seven years afterwards. I heard of him from time to time, but did not see him in his last illness. He had no return of disease in the mouth, but was said to have died with "a hard lump in his stomach," having suffered from persistent vomiting and symptoms of intestinal irritation. I could not get any further particulars of the "hard lump," but presume it was a cancerous mass in the stomach. So far as the cancer in the mouth was concerned, I think we may consider that in both a cure had been realised. Unfortunately, recurrence is the more frequent termination, and when this recurrence takes place in the lymphatic glands, as it does in the majority of instances, and not within the cavity of the mouth, the

patient will suffer less, and the death is a less dreadful one.

In the early period of cancer, as I have said, there is often difficulty in discriminating it from some forms of syphilitic affection of the tongue. There is difficulty, too, in saying when a warty, papillomatous growth becomes transformed into a malignant one. It is just possible that there might be a little hesitation in deciding whether a primary chancre was a carcinoma, because the chancre might be very hard, and perhaps otherwise resemble carcinoma; but a chancre would be on the tip of the tongue, probably in a young person, and associated with concurrent enlargement of the glands below the jaw, whereas in the early period of cancer, enlargement of glands would not occur. Moreover, if it be chancre other evidences of syphilis may be present, such as a rash on the skin or a sore throat; so that I do not think a chancre on the tip of the tongue of a young man or young woman should be difficult to recognise, the more so as cancer is almost unknown under thirty years of age. The same may be said with regard to the secondary manifestations of syphilis. There is not much likelihood of confusion, the mucous patches being the only possible source of difficulty; yet there are so many differences that I need not now detail them. I will only mention some of the features which distinguish gumma from carcinoma.

In tertiary syphilis you often find more than one gumma in the tongue at the same time, and the organ otherwise may show evidence of old syphilitic disease, such as the deep fissures I have already described, though these do not help much, because such a condition of the tongue is one of the predisposing causes of cancer. A carcinoma of the tongue is always a single lump. One or two instances have been recorded of cancer developing in two places on the tongue, an occurrence so exceedingly rare that for practical purposes we may exclude it. Then we have a difference of situation; the predominant place of selection in syphilis is the central portion and dorsum of the tongue, whereas the seat of predilection for the development of a carcinoma is certainly the border. Then the two disorders present differences in the type of ulceration. In a gummatous ulcer there is a certain amount of induration of an inflammatory kind, but not to any marked degree, which contrasts

sharply with the extreme hardness of the base and edges of carcinoma. In the latter you find the heaped-up, everted, or rolled-out edges and margins, whereas the gummatous ulcer shows thin and undermined edges. There is but little pain or salivation in the one, and much in the other. Still there are a certain number of cases where, in spite of all your acumen, you may not be able to discriminate with certainty. I would urge you, nevertheless, not to postpone effective remedial measures whilst you are determining the result of anti-syphilitic treatment. If this treatment has no effect upon the disease, you will have meanwhile sacrificed a precious interval of time, which may rob the patient of his chance of relief by operation. It is a common thing to say, "We will give the patient a chance of its being syphilitic rather than carcinomatous disease." But, in the excellent words of Mr. Butlin, "that means giving the carcinoma a chance"—a chance of killing the patient. You thus deprive the patient of his chance of recovery, and give the disease a chance of working its wicked will. In doubtful cases, the best course is to examine microscopically a portion of the growth.

Before referring to details of operative treatment, I may say a word regarding cases which we do not consider suitable for such a measure.

Operation, I fear, must be contra-indicated where the tongue is completely bound down to the floor of the mouth, because this indicates that the muscular tissue of the tongue is infiltrated by the disease; it also shows that there is, in all probability, a large amount of lymphatic gland implicated. Again, where the invasion of the lymphatic glands is so extensive that the disease cannot be effectively dealt with by local removal, operative interference should not be attempted; also in cases where it is very far back, involving the lower jaw or implicating the upper part of the larynx, and when the patient is the subject of very extensive disease, and weakened by loss of blood and sleep, he will frequently succumb to the immediate effects of the operation. Neither is any good done by operation on those cases where the carotid artery and pneumogastric nerve are implicated, or the jugular vein involved. You must, under these circumstances, employ palliative treatment. You can do much to mitigate the suffering by removing the teeth,—not merely diseased teeth but all the

teeth. The lingual nerve can be divided, which will give relief from pain and diminish the amount of salivation. Of course you may paint the surface with cocaine, but that is very temporary in its effect. With regard to the palliative removal of the diseased tongue, to take away the mass of horrible ulceration from the interior of the mouth, the idea is no doubt good; but in those cases where complete removal of the disease is not possible, operative measures cannot be successfully carried out. The whole disorder cannot be removed, epithelial disease remains in the raw surface of the stump, and the condition of the patient is in no essential manner improved.

There is one feature common to operations in this region, that they necessarily expose the patient to the risks of septic poisoning. The mouth swarms with micro-organisms, and the wound is therefore liable to infection. We find that septicaemia, septic pneumonia, and conditions of that kind are not uncommon. An operation on the mammary gland, far more extensive in its nature than that on the tongue, including the opening up of the cellular tissue in the axilla, need not expose the patient to any appreciable risk, and recovery will ensue without any rise of temperature and with no greater discomfort than that occasioned by the dressings and bandaging. In the mouth sepsis may take place in spite of our efforts.

Some modern methods are specially directed towards obtaining an aseptic condition in the mouth, and a distinguished German surgeon, Professor Kocher, has devised an operation whose chief end is to prevent sepsis. These efforts and many attentions to detail have reduced the mortality to one half its former average. Taking slight and severe cases together, I am sure it was not less than 30 per cent. in former days, whereas it may now be regarded as from 10 to 15 per cent. There is now less dread of haemorrhage. Formerly the chief effort of the surgeon was directed to the control of haemorrhage. The ecraseur, both wire and galvanic, was very largely employed, the lower jaw divided, and the two halves turned aside to enable the floor of the mouth to be reached; and also because it afforded free access to the lingual arteries. We now think so little of haemorrhage that the operation most frequently performed at the present time is that suggested by Mr. Whitehead, of Manchester, who

clips the disease away with a pair of scissors. In this the jaws are fully separated by a gag, the tongue is drawn out by a thick silk ligature passed an inch from the tip, with which steady traction is made forwards and upwards. The mucous membrane and the attachments of the tongue to the inner side of the lower jaw are cut through, which allows the tongue to be fully drawn out of the mouth. The muscles of the base of the tongue are then cut through from above downwards, as close to the hyoid bone as may be necessary. The vessels are seized and tied as they are divided, and a loop of silk is passed on each side through the remains of the glosso-epiglottic folds of mucous membrane, which enables the surgeon to draw forward the root of the tongue in cases of recurrent haemorrhage. This silk loop should, as a rule, be removed next day. If one half of the tongue only is to be excised, the thick mucous membrane of the dorsum is divided along the middle line, and the two halves of the tongue torn asunder—a procedure attended by little or no haemorrhage; then the affected half, after division of the mucous membrane and muscles connecting it with the jaw, can be pulled right out of the mouth and removed, taking care to divide the tissues far enough behind the growth, close up if necessary to the hyoid bone. In cases where the whole tongue requires to be removed, this method may be also adopted with advantage. The second half is easily taken out subsequently. When the deeper part of the tongue is reached the lingual arteries are either tied or twisted. If there should be any troublesome haemorrhage, pass your finger behind the hyoid and pull the stump and the hyoid bone forward; the haemorrhage is thus controlled and the bleeding point can be seized. This hint is Mr. Heath's, and it is an exceedingly valuable one where there is considerable bleeding.

The only other method I shall mention is that Professor Kocher has devised. A preliminary tracheotomy with pharyngeal tamponade having been first performed, he makes an incision commencing a little below the tip of the ear, along the anterior border of the sterno-mastoid, to the middle of the muscle or a point opposite the hyoid bone, thence forward along the upper border of the hyoid, and then along the anterior belly of the digastric to the chin. The skin and subcutaneous tissue are turned up as a flap, which

can be stitched to the cheek to keep it out of the way. The facial artery is ligatured and divided, then the submaxillary fossa is cleared out, including the submaxillary, sublingual, and lymphatic glands. The lingual artery is tied, preferably, before it passes beneath the hyoglossus muscle. The mucous membrane should be divided close to the jaw, when the entire tongue can be made to project through the wound, as much of the mylo-hyoid muscle as may be necessary being first divided. When the whole tongue is removed, the opposite lingual artery can be secured through a special incision. The tongue may be cut off with scissors, or, as Kocher prefers, with the galvano-cautery. This operation is very thorough, and diminishes the risks of sepsis and of recurrence. When the tongue is deeply invaded, although apparently only on one side, it will generally be necessary to remove the entire organ as well as all involved glands. Even when the disease is superficial, and affects the anterior part of the tongue, Kocher considers it safer to remove the nearest lymphatic glands, whether they appear on external examination to be implicated or not. I have not performed Kocher's operation, because I have not had a case in which it was necessary. Usually, I have split open the cheek from the mouth to the anterior border of the masseter muscle, which gives the operator very ample room; and where the tongue only has to be removed this method is sufficient, and the operation is completed subsequently by cutting through the structures I have mentioned with scissors.

I will only say, on the subject of after-treatment, that I have found the patient is able to swallow by the mouth in a surprisingly short time, and should be fed that way in preference to any other. If fluid food be given, and the feeding carried out cautiously, patients are able to swallow readily. Rectal enemata are practically useless, and the stomach-tube is very distressing. Feeding is one of the most important matters in the after-treatment, because you must remember that the patient is probably exhausted by the pain, loss of sleep, and foul discharge. Patients should generally be made to get up after two or three days. The mouth should be kept pure by frequent washing out with weak solution of permanganate of potash. The power of articulate speech soon returns, and except for certain letters, the absence

of the tongue is scarcely noticeable, and speech as a rule is better after total than after a one-sided removal of the organ.

A CLINICAL LECTURE
ON A CASE OF
MUSCULAR ATROPHY.

Delivered at University College Hospital,
April 16, 1896,

By J. ROSE BRADFORD, M.D., F.R.S.,
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GENTLEMEN.—The patient before you is a man æt. 37. Two months ago he first noticed numbness of the inner side of the right hand, and he presented himself in the out-patient room on March 9th. The numbness gradually spread to the elbow, and a similar condition was felt in the toes of the right foot. The patient also noticed a nervous shaking of the muscles of the right thigh when he began to walk. There was some wasting of the right hand, but the patient was not aware of it until it was demonstrated to him in the out-patient room.

The case has not yet been thoroughly examined, so that the diagnosis must be more or less provisional. It is clearly a case of muscular atrophy, and I want to make a few remarks on the varieties of muscular atrophy and the means of diagnosing them.

As regards the man's hand, you will notice a most marked wasting in the first interosseous space, as shown by the prominence of the metacarpal bones. There is not so much wasting on the palmar surface, but there is some which shows itself by the crinkling of the skin and the comparative flatness of the palm. When endeavouring to detect wasting, it is necessary to inspect the opposite hand, which serves to eliminate a number of diseases at once. You see there is a certain amount of atrophy of the other hand, but not in anything like the same degree as in the one we first examined, yet the atrophy is very similar as regards its distribution. Thus, the first obvious symptom of this case is that of

muscular atrophy, involving the small muscles of both hands.

To discuss the subject systematically, there are four kinds of muscular atrophy, or muscular atrophy due to four different sets of diseases. (1) There is the atrophy produced by disease of the brain; (2) Atrophy produced by disease of the cord; (3) Atrophy produced by disease of the nerves; (4) Atrophy produced by disease of the muscles themselves. The diagnosis between these four classes is mainly based upon the distribution of the atrophy. A man does not get wasting of the muscles of both hands from cerebral disease; you may sometimes see, in old cases of hemiplegia, as much wasting as in this patient, but you must remember that in certain cases of hemiplegia, particularly hemiplegia in the young, there is considerable muscular wasting, but not in both hands. Another vital point of distinction is, that muscular atrophy of cerebral origin is always associated with a considerable amount of paralysis; the palsy is more obvious than the wasting. For instance, a patient with atrophy from a cerebral lesion will not have such extensive wasting of the hand muscles as this, without general palsy of the arm muscles.

We will consider the muscles next. The case clearly is not one of pseudo-hypertrophic or idiopathic muscular atrophy; the latter diseases do not begin as this one has, nor in the same position. Again, the atrophy in those diseases is symmetrical and equal; here it is symmetrical and unequal.

We now pass to the important matter of *cord versus nerves*, and the points to be relied upon in attempting to make a diagnosis. Well, first we can eliminate peripheral nerves, because you know that the nervous supply of this interosseous muscle is different from the nervous supply of this thumb muscle. But it is very important to remember that you may get atrophy of this description from involvement of nerve roots. For instance, if a patient has a mediastinal tumour, or if he has phthisis, and his first dorsal nerve becomes involved as it passes over the first rib, he will get a wasting similar to this. There was a patient here not long ago who had such wasting, and it was extremely difficult, indeed almost impossible, to say whether the wasting was due to a cord lesion or due to a growth inside the thorax, involving the first dorsal root. So that, although

you can eliminate peripheral nerves as a cause of this kind of atrophy, you cannot eliminate, by looking at one hand, the nerve roots ; the first dorsal root will produce palsy exactly like this. But of course both first dorsal roots are not likely to be involved. So that the fact that the wasting is more or less symmetrical always suggests a cord origin, yet you must understand that this distribution in any one hand is quite compatible with a root lesion. In this case you either have to do with a cord lesion at the level of the first dorsal nerve, or with a lesion of the first dorsal nerve itself.

How are the two to be distinguished? Partly by the history, and partly by the present state. If a patient has a lesion of his first dorsal nerve on one side, as produced by mediastinal tumour, or a lesion of both his dorsal nerves, as might be produced by pachymeningitis, or by caries of the spine, he will have suffered very considerable pain ; not only that, but he will have probably suffered from shooting pain, confined to the track of the first dorsal nerve. The cutaneous distribution of the first dorsal nerve is along a strip of skin coming down on the inside of the forearm, and as far down as the styloid process of the ulna ; so that you would find pain shooting down the arm, accompanied by muscular wasting in this particular region. That would be very characteristic of a lesion involving the first dorsal nerve.

Patients with lesions in the grey matter of the cord, whether dependent on progressive muscular atrophy or on syringomyelia, or any other kindred disease, may have pain of considerable severity. Subjects of progressive muscular atrophy often have considerable pain, but it is an aching pain, most marked around the joints ; not a shooting pain confined to a definite area. That is a very vital point of distinction. If the patient has a lesion of the grey matter causing muscular atrophy, he may have no pain at all. On the other hand, he may have pain, and if that is an aching pain, which he calls rheumatism, it is very likely that his doctor will also call it rheumatism if such pain is felt in the vicinity of his joints. It is a great mistake to suppose that progressive muscular atrophy does not cause pain, just as it is wrong to say that poliomyelitis does not cause pain. Poliomyelitis sometimes causes so much pain as

to be mistaken for acute rheumatism. It is true to say that neither of these affections causes pain which is comparable to the pain of pachymeningitis ; but the bus driver who was in here last year with typical syringomyelia had very severe pain, so severe that a physician under whose care he had been diagnosed pachymeningitis. Apparently this man has not had very much pain. I should also mention that the subjects of syringomyelia may have numbness and tingling as well as pain.

The next point is, that if the patient have disease of the nerve roots, the probability is that a more or less extensive area of the cord will be affected by the disease of the meninges or disease of the bones, and as a result he may have pain over a wide area ; for instance, a man with muscular atrophy like this, dependent on cervical pachymeningitis, may have pain as high as the top of his head, simply because the great occipital nerve goes as high as that. The meninges will be thickened throughout the cervical cord, and pain may be produced, not only in the area of the palsy, but in other areas, which is a point of considerable practical importance. But the point on which most stress must be laid is the way in which the disease spreads. Disease beginning in the grey matter of the cord always spreads in a particular order. If the disease be outside the cord it is obvious that it need not spread in any particular order ; its spread depends upon where and what the particular disease is.

It is a fundamental law in progressive muscular atrophy, due to disease of the cord, that it always begins at the extremities of the grey matter in the cervical enlargement, not in the middle. Muscular atrophy always begins in the muscles of the hands or of the shoulder, not in those of the back of the arm. Further than that, if you have a patient with extensive wasting of the triceps muscle without extensive wasting of the biceps, if he has wasting of the muscles of the back of the arm out of proportion to the wasting of the other arm muscles, it is in favour of root disease. That is an empiricism ; and the only reason one can give is that one set of muscles are supplied from the middle of the cervical enlargement and the others from the extremities ; and that spinal morbid processes, for some peculiar reason, are liable to begin in the extremities, not in the

middle. Caries may begin in such a way as to involve the 7th cervical when degenerative diseases may not. The order of spread is also a great point.

If you make up your mind that the muscular atrophy is due to a cord lesion, and it is a slow process, as in this patient, there are many diseases which it can be. It may be progressive muscular atrophy ; it may be what is spoken of as amyotrophic lateral sclerosis, or it may be syringomyelia. If the condition be due to a root lesion you have to deal with pachymeningitis produced either by diseases of the bones, or by growths in the bones, or by syphilis, or by traumatism.

Now, how do you attempt to diagnose these various states ? First of all, as regards the difference between progressive muscular atrophy and amyotrophic lateral sclerosis, the points of distinction are as follows :—Amyotrophic lateral sclerosis is usually acute in its course and more rapid in its onset, but these rules are liable to exceptions. Secondly, patients with this disease have more pain ; yet I do not lay most stress upon this. Again, if there is a very great involvement of one muscle out of proportion to the other muscles, it is not likely to be amyotrophic lateral sclerosis. The next point is that sometimes patients with amyotrophic lateral sclerosis have a great increase in all their deep reflexes ; they may also have a very great amount of palsy. Patients with progressive muscular atrophy have not much palsy apart from the wasting ; that is to say, they have not a condition of extreme paralysis followed by wasting, but as long as they have any muscle left they have the power of movement, though, of course, that power may be very slight. The explanation of the great degree of palsy in amyotrophic lateral sclerosis is that the muscular wasting is very much more general in its distribution. So that, on casual inspection, I should not have thought it was a case of amyotrophic lateral sclerosis, because there is too much wasting between the thumb and first finger and too little wasting of the arm.

Now we come to syringomyelia. This is a fairly common cause of muscular atrophy, occurring, perhaps, in about 30 per cent. of the cases of so-called muscular atrophy. Syringomyelia is not a disease, but is a term given to a particular group of symptoms produced by a destructive

disease of the grey matter of the cord, and more particularly of the cervical enlargement. To put the matter crudely, progressive muscular atrophy is a destructive affection of the anterior cornua, while syringomyelia is a destructive process in both anterior and posterior cornua, all four are affected.

As regards the phenomena of syringomyelia, patients present themselves with various symptoms—some with muscular wasting, as this man did ; some with paralytic symptoms, and it is possible that the patient in the next bed may be such a case. Other patients come for sensory symptoms ; one man I know had the typical incident of burning his fingers without knowing it. Another man, whose case I quoted to you a few days ago, first became aware of something abnormal by the fact that when scrubbing himself in his bath he did not feel the brush. Patients may come on account of weakness of the extremities, and we may regard the occupant of the next bed as an illustration of this. Again, they may come with painless whitlows. Another disease such patients may present, though I have not seen more than one example for several years, is perforating ulcers of the soles of the feet. So important is this sign, that, when accompanied by excess of knee-jerk, syringomyelia may be safely diagnosed. Lastly, there is a curious group of cases in which the patient comes for advice for joint symptoms, such cases as a casual person might diagnose as rheumatoid arthritis. Such a case was so diagnosed recently ; but that patient volunteered the statement that he ran a nail through his foot without feeling it. He was very much deformed, and in the absence of a careful examination one might easily have concluded that it was a case of bad rheumatoid arthritis. A very well-known case went the round of the London hospitals and was diagnosed as rheumatoid arthritis, but in a parish infirmary it was found to be syringomyelia. Occasionally Charcot's joints are seen in syringomyelia, but I have not seen this myself. So you see it is a very protean malady, and, in order to impress them upon you, I will repeat the symptoms with which patients may come to you :—(1) They may have muscular wasting ; (2) Weakness of the extremities ; (3) Sensory disturbances, such as painless whitlows, burning of the fingers without being aware of it,

which is common ; (4) Perforating ulcers, which are not very rare ; (5) Joint symptoms of the type of rheumatoid arthritis, but not running the same course.

As to diagnosis, this is generally fairly easy, and where the diagnosis is not made it is usually because this disease is not looked for. Syringomyelia is diagnosed mainly by the characteristic sensory loss. The text-book statement is that you get loss to heat and cold, loss to pain, and persistence of sensation to touch. That is all very well, and if you have a very marked case, such as the man in Ward 7, about whom I have already spoken, you find that. But there are certain other characteristics about the sensory loss which will help you.

First, as to its distribution. The distribution of the sensory symptoms is one of the most extraordinary features about syringomyelia. This shows that the sensory loss in this disease is not due to impairment of conduction. If a patient has syringomyelia in the cervical enlargement of his cord, you will not find any sensory loss in his legs, though you may find a little loss on his trunk ; but the loss is found to be vest-like in its distribution, *i.e.* it involves that area which is covered by a vest. It may extend to the costal margin, and it involves the arms. Further, it may involve only half the body, but the vest-like distribution in that half is preserved. If you think of that you will see that it is very remarkable. Suppose the lesion is in the cervical cord, it does not interfere with conduction from the parts below and so produce analgesia in this region of the body, but the disease destroys the antero-posterior cornu, and destroys the sensory fibres as they enter the cord.

The analgesia terminates by a sharp line, which is a very important point. The patient may have analgesia corresponding to the part covered by a glove, or by a gauntlet, or it may be even higher. But wherever the analgesia is, it involves both surfaces of the limbs, and terminates in a sharp line, which is a condition not met with in analgesia produced by a root lesion. The roots are distributed in strips down the limb. The only disease which is said to produce similar symptoms is leprosy neuritis, but there is some doubt about that. The third characteristic of the analgesia produced by syringomyelia is that which the text-

books put first, viz. diminution to pain and diminution to heat and cold. But, as I said, you must not expect to get absolute loss to heat and cold : in a slight case the only evidence of analgesia may be the fact that boiling water in contact with the skin is felt as only warm water, or when warm water is placed next the skin, the patient is unable to say whether it is warm or cold. Again, some cases manifest only loss to heat or loss to cold, but not both. Then of course in a very extreme case, when the destruction to the grey matter is complete, there is loss to pain. I suppose you all know why this is ; the posterior root, as it goes into the cord, divides into two bundles, one of which goes into the grey matter and the other into the white matter. In syringomyelia, the bundle which goes into the white matter does not usually get involved, and it conducts touch impressions, whereas the bundle which goes into the grey matter becomes involved, and does not convey the impressions which go through it in the normal state, namely, those of pain, of heat, and cold. For teaching purposes, one always draws attention to three diseases : syringomyelia, tabes, and one which I am afraid you do not often see—ataxic paraplegia. In tabes, the posterior roots are affected, they waste and undergo certain changes ; in tabes also the patient has sensory loss of all forms—touch, pain, heat, cold and muscular sense—because his posterior roots go. A patient with syringomyelia only loses pain and heat and cold, because his grey matter goes and his white matter remains intact,—the posterior roots themselves are not usually affected. In atactic paraplegia the opposite condition obtains, the grey matter is not affected, while the white matter is affected. Such a patient loses muscular sense, but he does not lose the power of appreciating pain or heat or cold.

The other physical signs met with in syringomyelia are rather interesting. First of all, certain other muscles, besides the voluntary, are affected, more particularly the muscles of Müller, so that such patients very often have unequal palpebral fissures, or narrow fissures on both sides. They get the sympathetic involved because the fibres which are distributed to the muscle of Müller come out, roughly, at the first, second, and third dorsal nerves, and if the grey matter of that region is affected, not only do you get wasting of the

intrinsic muscles of the hand, but also involvement of certain ocular muscles. There was a beautiful illustration of that in the case I quoted before, of a man who had wasting of those muscles of one side, and paralysis of the muscle of Müller, with narrowing of the fissure on the same side. Unless you are aware of the normal condition before the illness, it is difficult to detect it if on both sides. Sometimes the pupil is involved; it is not sufficient to try whether the pupil contracts in the presence of a strong light, it is also necessary to ascertain whether it expands in relative darkness. There is another symptom which, as far as I know, all advanced cases exhibit, namely, a very remarkable sweating. This was shown by the man in Ward 7. The sweating is probably due to paralysis of the sweat nerves, just as the muscular wasting is caused by paralysis of the voluntary nerves; and this sweating has the same distribution as the anaesthesia, and will sometimes terminate along a mathematical line. Those of you who saw that other patient will remember that he sweated, roughly, down to his tenth dorsal, and the whole of his body above that point was covered with perspiration, while below it was absolutely dry. I do not know any disease of the cord which does that except syringomyelia. Sweating of the face is sometimes caused by an aneurysm pressing on the sympathetic. Besides the symptoms which I have mentioned as being met with in syringomyelia, there is spontaneous fracture, and another, which is important, spinal curvature. Patients with syringomyelia frequently have spinal curvature; it is doubtful whether it is present in this patient. I cannot now go into the question of how it is brought about, because there is a considerable amount of dispute about it. Still, there is no doubt about its occurrence, and it is as marked as in Friedreich's disease. In this particular case the diagnosis would lie between syringomyelia and progressive muscular atrophy, and would depend upon examination of the cutaneous sensation. I was told he had complete loss to pain in the distribution of the first dorsal; if so, it would be syringomyelia; if not, it would be progressive muscular atrophy.

As regards the diseases which produce syringomyelia, the one of most importance is a tumour, a glioma. Such cases run a fairly acute course; the glioma grows into the anterior cornu, producing

these motor symptoms, or grows into the posterior cornu and produces sensory and trophic symptoms. Every now and then one meets with cases of degeneration like progressive muscular atrophy, affecting both anterior and posterior cornua, which are called cases of syringomyelia, but I do not think those cases are very common. Most of the acute cases which have been published were due to growths. Myelitis affects sometimes not only the anterior cornu, but spreads into the posterior, so that the pathological condition produced is a kind of big central canal from which the grey matter drops out.

Lastly, there are the congenital cases, which some people call hydromyelia, in which there is imperfect development of the grey matter, associated with imperfect closure of the spinal canal.

The only other point which I can demonstrate in this patient is the state of his deep reflexes. The knee-jerk is greater on the right side, and he also has clonus. That does not exclude pachymeningitis, but it makes it very improbable. If a man has pachymeningitis, producing this condition of things, it is extremely unlikely that it would make one knee-jerk greater than the other. Pressure on the cord would be fairly equal, and both knee-jerks would be affected in about the same degree. If muscular atrophy and the knee-jerk are more marked on one side though present in both, you are justified in diagnosing a lesion of the cord, and I think the lesion is a spinal one in this case. There is one stock thing which one ought perhaps to say: Progressive muscular atrophy is not chronic inflammation of the anterior cornu; progressive muscular atrophy is not chronic poliomyelitis. One knows, clinically, that it is not chronic poliomyelitis, for this simple reason: as far as I know, nobody ever saw an inflammatory lesion in the spinal cord beginning first on one side, and then, after a certain interval, affecting precisely the same spot on the other side. That is not the method which an inflammation follows; inflammations are very random in their distribution, and if you see acute poliomyelitis, it is very exceptional to observe similar muscles on the two sides of the body affected to equal extent. Another reason why progressive muscular atrophy is not chronic poliomyelitis is a pathological one. In every case of progressive muscular atrophy there is degenera-

tion of the lateral column above the level of the lesion. If a patient has a lesion of the cervical enlargement, producing atrophy of the muscles of the hand in the way shown here, and you were to examine the medulla, you would find degeneration of his pyramids. It is perfectly clear that you cannot assume that the degeneration in the pyramids is secondary to the loss of the grey matter. Recently it has been found that in progressive muscular atrophy and in amyotrophic lateral sclerosis, if the cortex cerebri is examined, the nerve cells have undergone degeneration. Progressive muscular atrophy, as one always teaches, is one of the most remarkable diseases, from a pathological point of view, because it is a system degeneration of the whole motor tract, and you have no more right to say that it begins in the cord than that it begins in the thumb muscles. This is not a matter of mere theoretical interest, but is of very considerable importance, because you may make a wrong diagnosis between progressive muscular atrophy and chronic poliomyelitis : there is such a disease as the latter, and it is not very uncommon. Chronic poliomyelitis does not progress, which is the most characteristic feature of progressive muscular atrophy, and in this it resembles most degenerations.

I cannot go into differential diagnosis now, but the failure to distinguish those diseases is a mistake which men get very grave discredit about. They see a case of wasting such as this (assuming this to be unilateral), and nine out of ten probably say it is progressive muscular atrophy at once, whereas you must remember that poliomyelitis may do this. The man I have mentioned several times before was probably a case of chronic poliomyelitis ; he had worked in lead. I have seen several cases of this after typhoid fever, one of which made a particular impression on my mind ; it was a woman who had been told she had progressive muscular atrophy, and the statement of that opinion produced rather serious results. It was proved not to be progressive muscular atrophy, which, you must remember, is a far more serious disease than poliomyelitis, and far less amenable to treatment.

As regards differential diagnosis, poliomyelitis probably only affects one side ; progressive muscular atrophy always affects both sides, and the interval between the involvement of the

second side is usually a few weeks, and at the outside is never more than six months ; it is much more likely to be only five or six weeks. That is one of the fundamental points of distinction.

A pertinent feature as regards the etiology is that poliomyelitis is frequently toxic, or it is the result of an acute specific, whereas nobody knows the cause of progressive muscular atrophy with any degree of accuracy. But it is not due to toxic causes, and it does not follow acute specifics.

The only treatment in these cases of syringomyelia is the hypodermic administration of strychnia, commencing with 1*m* and going on to 5 or 10 ; but 10*m* of liquor strychnia is a very big dose, and cannot be given until the patient has got accustomed to the drug.

THE APPLICATION OF HERNIAL TRUSSES.

Demonstration given before the Dublin University Biological Association

By G. JAMESON JOHNSTON, M.A., M.B.,
Vice-President of the Association,
Assistant Surgeon, Richmond Hospital.

MR. PRESIDENT AND GENTLEMEN,—I have been led to the selection of this particular subject through the following reasons : The small and indefinite nature of the information concerning trusses given in the ordinary text-books ;* the fact that the boom in operations for radical cure has diminished the opportunities of students for studying the application of trusses ; and, as you all cannot hope to become hospital surgeons, and may be placed in circumstances where an operation is out of the question, I thought the following remarks might be of some practical advantage.

A good many surgeons merely content themselves with taking an approximate measurement round the pelvis, order a truss, and leave the rest to the instrument maker. This is hardly sufficient nowadays ; though fitting of trusses is not so attractive as operating for radical cure, still, an interest may be worked up in it sufficient to over-

* The second volume of "Treve's System of Surgery," just issued, contains much useful information concerning trusses and their application.

come the difficulties, which are more apparent than real. Every medical man ought to be able to take proper measurements, know the exact size necessary, shape of the pad, and best position for placing it, also the shape and strength of spring required for each case, and ought to be able not only to recognise a badly-fitting truss, but also the particular points in which it is defective.

First, then, in general terms, what is a truss? A truss is a pad held in position by a spring or belt, or combination of both, by means of which a weak part of the abdominal wall (or elsewhere) is rendered sufficiently strong to prevent the extrusion of contents, or by means of which irreducible viscera are protected and prevented from further extrusion.

Trusses might be conveniently classed as ordinary and special. The ordinary forms are inguinal, femoral, and umbilical, of which the two former may be single or double; it is mainly to the inguinal that I wish to refer. The special are obturator, lumbar, scrotal, &c., and are dismissed by merely mentioning them.

Trusses vary in themselves in the materials of which they are composed and in the shape of the spring and constitution of the pad. I shall first describe the ordinary spiral spring, or so-called circular spring. I say so-called, as the spring is usually only two-thirds of a circle.

The essential parts are the pad, which is placed on the parts requiring support, and the spring and belt combination, which fixes the pad and provides the power of resisting extrusion of viscera or solid organs; usually there is also a thigh strap for further fixation of the pad during movements of the body.

A good truss should be light, firm, and elastic, of just sufficient strength to keep the hernia reduced in all positions of the body, and under all circumstances of ordinary exertion; if too slight or badly fitting, it allows the hernia to come down behind it; if too strong it may cause pressure absorption and thus enlarge the opening, or it may cause inflammation of irreducible contents.

The pad is commonly made of horsehair, covered with leather, but various other materials are used: solid rubber, wood, air, glycerine, water, &c. The solid ones are probably best, as they are more secure. The surface should not be too convex, as it may then cause pain and enlarge the aperture.

The shape of the pad is pyriform, with the broad end down; the upper edge should be continuous with the down slope of the spring. The angle downwards varies with the rupture, being practically vertical in femoral rupture. The pad is preferably immovably fixed to the spring, it should not touch the pubes when fitted; if necessary to have it low down it should be cut out so as to prevent pressure against the bone. The pad should extend over the aperture at least half an inch all round, and more in large herniæ. There are two studs on the outer surface of the pad, the upper for the cross strap and the lower for the thigh strap. The position of the pad is of the greatest importance. In ordinary oblique inguinal herniæ it ought to be placed on the internal ring and canal; this is one point which must be seen to, as patients usually cannot see why it is not placed over the "lump," and often act up to their inclinations by changing the position of the pad. The pad is directed back, slightly outwards and upwards, in thin people; in fat people with large abdomens the upward inclination must be increased. In direct hernia the pad is placed over the external ring; in femoral the pad is directed back over the femoral canal at the level of Gimbernat's ligament.

These points about the pad may be summed up in saying: Place the pad over the point of escape from the abdominal cavity and see that the pressure is in the proper direction.

There are three types of spring: the German, which is very rigid, not elastic, fits exactly the outline of the body at rest, scarcely presses at all when the abdomen is relaxed, but offers great resistance to abdominal expansion.

The French, which is light, very elastic and clinging, always pressing even when at rest, it is uncomfortable and may cause pressure absorption. It is rather curious how, even in such things as truss springs, the characteristic feature of these two nations is reproduced, the German rigidity as contrasted with the French flexibility.

The English type is an intermediate condition. The spring encircles two-thirds of the pelvis, and the circle is completed by the cross strap, which is continued from the end of the spring to the upper of the two studs on the pad. The spring passes round the pelvis just below the iliac crests; if placed above this level, the contraction of the abdominal muscles displaces the truss; if placed

on the crest the skin is chafed ; if placed too low the gluteal muscles, in contracting, are liable to change the position of the truss. The strength of the spring ought to vary with the difficulty or otherwise in maintaining reduction, and also with the occupation of the wearer, a stronger one being required for a blacksmith than a tailor. The presence of chronic cough will necessitate a stronger spring.

The function of the thigh strap is, as I have said, to prevent displacement during the movements of the body, and should pass from just behind the bend of the shoulder along the gluteal fold through the perineum to the lower of the two studs on the pad. It should just feel tight when the patient stands up.

There are some particular patterns of truss which it is necessary to notice, as they are often used by the better classes ; they are mostly expired patents, and in nearly all the objection holds good that they sacrifice efficiency for comfort.

The principal is Cole's, which has a spiral spring inside the pad, in addition to the ordinary spring ; this probably lessens the stability of the pad. It requires no thigh strap, and may be useful, like most of the following, for slight hernia and for patients who are not subject to much exertion.

Salmon and Ody's : the spring is self-retaining, has one pad behind at centre of back, another in front across mid-line, hence called the "opposite-sided" truss. The front pad is articulated by means of a ball and socket joint, allowing closer application in the various movements of the body.

The Moc-Main lever truss is more comfortable than safe, the spring is in the pad itself, there is none in the belt. Its non-elasticity is a serious defect, as it is sometimes too loose and at other times the reverse, and cannot press equally in all the required directions.

Wood's pads are solid and have a notch to save the cord from pressure : it is doubtful whether this is necessary or not ; it is a much better truss than those just mentioned.

In ordering a truss for inguinal or femoral hernia, send a measurement taken from the lower part of the hernial orifice up to the anterior superior spine of same side round pelvis, an inch or so below the crest of the ilium to the anterior superior spine of opposite side, finishing at the

upper part of the hernial orifice, state the kind of hernia, the side, size of ring, strength of spring, and the age and sex of the patient.

In fitting, see that the hernia is reduced, note the position of the testicle, this latter point is especially important in children, and test by letting the patient strain and cough in that position in which the abdominal muscles are most relaxed, say with the knees and thighs flexed and the legs wide apart.

It is advisable to have a celluloid truss for bathing in, and a second truss ought to be kept to change with the ordinary one for the sake of cleanliness. A point always arises whether a patient ought to wear a truss at night or not. I advise patients to have a light one for this purpose, as one must always remember that, no matter how long a truss is worn, if once the hernia is allowed to come down, the commencement of the cure can only date from the last reduction. Dyspepsia often is the result of hernia, and this renders the patient restless at night ; getting up to make water, cough, and other trivial conditions are serious enough in this consideration. In any case, the truss ought to be taken off in the re-cumbent posture and replaced in same.

I have purposely avoided the question of what cases are suitable for treatment by truss, a "cure" can only be expected in children and young people. But in cases where the wearing of a truss is recommended, I think that an ordinary spiral spring truss properly fitted will give the best results. In children I have found a truss made of pure rubber and consisting of a band round pelvis with thigh-piece, having no spring, the pad being a rubber sac filled with air, is more efficient in every way than any other.

A Note on the Administration of Nitrous Oxide Gas with Oxygen for the Removal of Adenoid Growths.

By H. BELLAMY GARDNER, M.R.C.S., &c.
Assistant Anæsthetist to Charing Cross Hospital.

I HAVE found nitrous oxide gas, with a small percentage of oxygen, to be a very suitable anaesthetic for short operations. We have used it continually now for ten months in the Aural

Department of Charing Cross Hospital during the operation of removing post-nasal adenoid growths with the best results, especially in patients who have to bear a journey home very shortly afterwards.

The advantages I would claim for its use are :

- (1.) That it is not attended with danger to life.
- (2.) That no preparation for the operation is required.
- (3.) Haemorrhage is not affected by it.
- (4.) Jactitation and cyanosis produced by pure nitrous oxide are absent.
- (5.) Any position desired by the operator may be safely assumed.
- (6.) The available anaesthesia is 10 or 15 seconds longer than that yielded by gas alone.
- (7.) Unpleasant after effects of any kind are of very rare occurrence.

I hold that it is the anaesthetist's duty to render the patient unconscious of pain with as little damage to the physiological processes of his system as possible, and for these operations I find it a great improvement upon gas alone, while the danger of chloroform and the sickness of ether are entirely obviated by its employment.

Some patients will move a little, and in some there will be a little phonation, but you will find on questioning them that they felt nothing of the operation, and would quite willingly take the anaesthetic again. Now, this result is not often attainable with gas alone, for children yield a very short anaesthesia under its influence, and are often very blue and rigid, and oftener still have some bad dream, while if oxygen is present unpleasant dreams are quite uncommon, and there will be muscular relaxation with good normal colour.

It is well, if the little patient will permit you to do so, to insert a small Hewitt's mouth prop between the teeth before applying the face piece, in order to facilitate the introduction of a Mason's gag at the height of anaesthesia just behind it ; you can then remove the prop, and adjust the position of the head for the operator to begin ; with a little practice this last is only the work of a moment.

Artificial Feeding of Infants.—

B	Milk,			
Cream	aa	3j
Water,				
Lime water	aa	3ij
Malt sugar	3ss	
—HIRST.				

A NOTE FROM THE CLINIC

Of Mr. ANDREW CLARK,
Surgeon to Middlesex Hospital.

Diagnosis and Treatment of Perforating Ulcer.

THE man in this bed was admitted some months ago with what was supposed to be perforating ulcer of the foot ; on examination no nervous symptoms were to be discovered at all, and the case was treated as a soft corn, and the man left the hospital with the wound quite healed. He seems, however, to have neglected himself, and he now comes back again with an ulcer on the foot in the same place, with a long sinus extending from it, but still no nerve symptoms. The probability is that he will have to lose the front of his foot, the sinus shows no signs of healing, and carious bone can be felt at the bottom. A good many so-called perforating ulcers are only soft corns, and these from long neglect attack the bones and joints of the tarsus. Perforating ulcers occur in connection with tabes, spina-bifida, or syringomyelocele. In tabes many remedies have been recommended, among these I may mention iodide of potassium, mercury, arsenic, chloride of gold, and silver salts. According to some, if you stay the progress of locomotor ataxy with these drugs, then you may be able to cure the ulcer, others hold that electricity is the only means of cure. It is not right to entertain any idea of amputation till you have exhausted all other means. One point to be well observed is that though the patient does not feel pain in these cases, still rest must be obtained for the part by keeping the patient in bed. These ulcers may heal if treated merely with a stimulating application, such as resin ointment, and the same result will often follow cleanliness and prolonged rest. In regard to electrical treatment there are cases recorded, in which by using the continuous current for many months, healing of these ulcers has been brought about. A convenient way of employing it is to put one pole into a footbath, and let the other be held over the buttock in the course of the sciatic nerve.

It is important also to remove any carious bone, and with a sharp knife to clear away the overgrowth of cuticle at seat of the ulcer. In connection with

perforating ulcers, there may be also noticed a blistered condition of the sole, with great thickening of the skin, and from the trophic lesion there may be further noted that the hair becomes harsh and brittle, or herpes is manifested, and then under the cuticle ecchymosis occurs associated with much pain.

In some cases the nails decay and fall off, and the teeth also may become loose and fall out. It is very usual to find perforating ulcer commencing opposite the head of the first metatarsal as a corn; the discharge is usually thin and scanty, and the probe passed down the resulting sinus touches bare bone, which is found to be dry and spongy. Motor paralysis is not noticed with this condition, but the foot will be found to be more or less anaesthetic.

A great deal of attention has been drawn to the fact that the nerves leading to the affected part exhibit atrophy, and hypertrophy of the endoneurium, pointing in the direction that disease of the nerves leading to the degenerated part, or in the sensory column of the cord belonging to the foot, lead to an impairment in nutrition, and this is, therefore, deemed the primary cause of the disease. Some surgeons are in favour of the view that practically only sensory nerve fibres are involved in this complaint.

One observer believes that if there be double perforating ulcer, that is to say, a perforating ulcer on each foot, investigation should be at once undertaken with a view to exclude syringomyelia. A curious point in connection with perforating ulcers is the fact that cases have been recorded of spina-bifida undergoing spontaneous cure, and being followed by perforating ulcers in the feet, and partial paralysis of the lower limbs.

The practical outcome of our consideration, however, is that the sinuses in these cases should be carefully scraped, and the thickened cuticle should be removed, to facilitate which fomentations should be freely applied. Some have suggested that the wound if sluggish should be lightly treated with a thermo-cautery, or subjected to the action of the strongest nitric acid. Supposing, however, that no good results from all these measures, it becomes then a question of amputation, and care should be taken to operate above the level of the anaesthesia. In some cases recurrence has been known to occur in the stump.

REVIEWS.

Colour Vision and Colour Blindness. By J. E. JENNINGS, M.D. (F. A. Davis & Co.) Price \$1.00.

This little manual, on an exceedingly important but very special subject, seems to us to be carefully written and trustworthy. The tests proposed are certainly easy to carry out, and sufficiently exhaustive to detect defects in colour-sight in all in whom it exists. There are several equally good manuals written by English authors, but this is cheaper, and is likely therefore to be preferred.

Current from the Main. By W. S. HEDLEY, M.D. (Lewis & Co.) Price 2s. 6d.

This little brochure, of some thirty pages, is an endeavour to show and, having shown, to meet the dangers of using the electric lighting currents for purposes of medical treatment. As this current can only be used directly by those who have had it introduced into their houses, the work can only apply to a limited number. This number will probably soon increase, and we should advise anyone proposing to join it to read Dr. Hedley's work before doing so; he will thus learn what there is to seek to avoid and how to avoid it: prevention is much better than cure in such cases.

THERAPEUTICAL NOTES.

New Treatment of Burns.—Poggi and Vergely (*Medical Week*, vol. iv, p. 96, 1896). Dr. Poggi has found that the addition of a few teaspoonfuls of potassium nitrate to a bath, in which the burned part is incased or plunged, will quickly cause the cessation of pain. The water becomes heated after a while, and the pain reappears, but quickly subsides upon the addition of another quantity of the salt. When continued for several hours, it is said this method of treatment will prevent the production of phlyctenæ. Professor Vergely, of Bordeaux, obtained a similar result by covering the burned tissues by means of a paste prepared by mixing calcined magnesia with a certain quantity of water, and allowing it to dry upon the skin, and renewing it as soon as detached. The wounds are stated to heal satisfactorily, and pain is prevented.

The Treatment of Vulvar and Anal Pruritus.—The *Journal de Médecine de Paris* states that Morain carries out a treatment as follows: Morning and night the vulva is doused with water as hot as the patient can possibly stand, containing 1 per cent. of chloral, carbolic acid, or vinegar. Afterwards it is well to employ by means of a compress the following prescription: Rx Hydrochlorate of cocaine, 15 gr.; distilled water, 3 dr. Or the following ointment: Rx Menthol, 45 gr.; olive oil, 15 mins.; lanolin, 4 dr. Instead of this ointment the following may be used: Rx Bromide of potassium, salicylic acid, of each 15 gr.; glycerite of starch, 5 dr.; calomel, 2 gr.; extract of belladonna, 2 gr. Or the following lotion may be employed: Rx Bichloride of mercury, 30 gr.; alcohol, 3 dr.; rose-water, 10 dr.; distilled water, 1 pint. In still other cases it may be necessary to employ the continuous or interrupted electrical current to the part as strong as can be borne. In particularly rebellious cases resection of the parts may be absolutely necessary. In pruritus of the anus, Morain regards the influence of very hot water, repeated two or three times a day over the parts, as the best. He also suggests, with a view of overcoming any tendency to constipation, that the patient after preparing for bed shall use an injection of oil, or at least introduce within the anus some vaselin. An additional means of treatment of this troublesome disorder is to apply every night and morning a glyceroll made as follows: Rx Lime-water, 1 dr.; calomel, 30 gr.; glycerine, 5 dr. Or the following ointment: Rx Calomel, 1 dr.; vaselin, 1 oz. Or, Rx Red oxide of mercury, 1 dr.; vaselin, 1 oz. Or, again, absorbent cotton smeared with oxide of zinc ointment may be applied to the anal opening; or cauterization of the painful part with nitrate of silver in the proportion of one drachm to the ounce may be used. Finally, quadrilateral scarification of the skin about the anus may be necessary.

Hæmorrhoidal Flux.—If the hæmorrhage occur daily, is considerable, and the patient weak and nervous, it should at once be arrested. The writer has generally succeeded in suppressing the hæmorrhage in these cases and restoring the patient's health by strictly observing the following course: To anæmic patients of a nervous and serious temperament he has given the following tonic and anodyne pills: Rx Sulph. ferri, 1 dr.; ext. belladonnæ, 6 gr.; syr. simplicis, q. s. M. Fiat massa in pilulas xxiv dividenda. One pill morning, noon and night. Or, to fulfil the same indication, the following solution has been given to very weak, nervous, and anæmic patients, with the immoderate hæmorrhoidal flux: Rx

Ferri citratis, 4 dr.; sulph. quininæ, 1 dr.; acidi citrici, 30 gr.; aq. destillatæ, 1½ fluid ounces. Fiat solutio. From 20 to 30 drops of the solution should be taken three times daily in a wineglassful of the cold infusion of wild cherry bark (*Prunus Virginiana*); at the same time the infusion may be drunk freely through the day.

In addition to these it is highly important and necessary, in order to arrest the hæmorrhage, to inject into the rectum half a pint of cold water just before each evacuation of the bowels, and soon after the evacuation 2 or 3 ounces of lime water (*liquor calcis*), to be retained if possible.

When the hæmorrhage occurs guttatum from the mucous membrane of the rectum, the writer has frequently succeeded in arresting it by the application of nitric acid of a specific gravity of 1.500. The bleeding mucous surface should be delicately touched with it by means of a fine glass-hair brush or with a wooden spatula. Two or three applications will be sufficient, five days intervening between them. Great care should be observed to confine the application only to the affected bleeding mucous surface, and not let any of the acid come in contact with the fine, delicate, and highly sensitive muco-cutaneous tissue about the verge of the anus. Immediately after applying the acid, olive oil should be applied to somewhat modify its action. When the hæmorrhoidal flux is very profuse and escaping *per saltum* from organised tumours, nothing will succeed in arresting it but the complete removal of the tumours themselves, and it is much easier to arrest the bleeding and effect a complete cure than when the hæmorrhage issues guttatum from the whole mucous surface. In the former case there is almost always to be found the dilated extremity of a capillary tube in the tumour if regularly organised and of sufficient age. By removing the tumour or tumours the bleeding of course ceases by means of the plastic inflammation which follows the operation, and which completely seals the bleeding vessel which supplied it. In all such cases it is of the utmost importance, for obvious reasons, that the patient should have one regular and easy passage daily, avoiding as much as possible extra straining efforts. If the bowels are obstinately constipated, mild laxatives must be taken in addition to the enemata of cold water. The writer has frequently used the following aperient with advantage in such cases: Rx Sulphuris loti, magnesiae calcin, sacch. lactis, &c. 4 dr., M. fiat pulvis. Two teaspoonfuls of the powder should be taken whenever indicated. The writer has used balsam of copaiba and oil of turpentine with good results in the treatment of excessive hæmorrhoidal flux. Acidulated drinks and some of the light wines may be continually used with great advantage during the treatment, together with all means of prophylaxis.

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WEDNESDAY, SEPTEMBER 9, 1896.

A CLINICAL LECTURE ON CIRRHOSIS OF THE LIVER IN CHILDREN.

Delivered at the Victoria Hospital for Children, Chelsea,
June 4th, 1896,

By H. D. ROLLESTON,
M.A., M.D.Cantab., F.R.C.P.,

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Assistant Physician and Lecturer on Pathology,
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THOUGH somewhat rare, cirrhosis of the liver in children has recently, and especially in America, attracted considerable attention.

In the 'Encyclopædia of Children's Diseases,' Hatfield was able in 1889 to quote 156 recorded cases, including 61 collected by Dr. R. P. Howard, and since then a fair number are to be found scattered in medical literature. It is therefore by no means a medical or pathological curiosity, and I take this opportunity of considering somewhat briefly the various forms of cirrhosis which may occur in children, and shall refer to their causation and the clinical aspects they present.

In addition to the forms of cirrhosis met with in adults, children are liable to two special forms, of which one, the peculiar cirrhosis of congenital syphilis, is well known, while the other is less important, and not so often recognised,—the fibrotic change met with in rickets.

Since in these two forms of cirrhosis, which are special to children, the symptoms of the primary disease overshadow any that may be due to the hepatic change, it may be well to dismiss them before considering those varieties of cirrhosis which are of more clinical importance and which resemble on general terms the disease in adults both in bedside symptoms and anatomically.

LIVER OF CONGENITAL SYPHILIS.

This condition, which is the only true cirrhosis
Vol. VIII. No. 20.

resulting from syphilis, is met with in the hereditary and not in the acquired form of the disease. The acquired form may, it is true, give rise to gummatæ and to a local fibrosis, but not to a generalised interstitial hepatitis.

Morbid Anatomy.—There is a diffuse infiltration of the lobules themselves with young connective tissue, the individual liver cells being separated from each other, a condition which is described as a pericellular, monocellular, or perhaps better an intercellular or unicellular cirrhosis. The liver is enlarged, usually yellow in colour, and on section, especially in cases of some standing, small whitish spots appear at intervals. To the naked eye these look like small miliary tubercles, and microscopically they so far resemble them in being localised collections of small round cells. Tubercle may indeed be associated with intercellular cirrhosis, and the distinction between these small syphilitic and miliary tubercles depends on the absence of tubercle bacilli.

It is interesting to note that Adami* has recently described pericellular cirrhosis in the Pictou Cattle disease of Nova Scotia. The change is due to a minute bacillus, and gives rise, unlike congenital syphilitic disease of the liver, to ascites, diarrhoea and muscular weakness, in short to symptoms like those of ordinary cirrhosis.

Clinically the cirrhosis of congenital syphilis shows itself by no special signs or symptoms except that as far as we know it occurs in well-marked cases of the specific cachexia, and that the liver and spleen are enlarged. In connection with the enlargement of the liver it must be borne in mind that normally in young children the liver projects further down than in adults; this Henoch points out is not merely due to the relatively larger size of the organ in children, but also to the ribs being more horizontal and so leaving the organ more uncovered.

The cirrhosis of congenital syphilis should be considered as belonging to the secondary stage, and is therefore a condition from which recovery

* Middleton-Goldsmith Lecture, 1896.

or resolution can take place. This consideration is of great interest in connection with the question as to the ultimate state of the liver in a patient who has suffered from pericellular cirrhosis. In some cases of congenital syphilis *gummata* have been found associated with pericellular cirrhosis, but it is very rare. In such instances the secondary manifestations have been succeeded by the tertiary. But apart from this it appears that pericellular cirrhosis is a transitory and curable condition. At any rate, the distribution of ordinary cirrhosis occurring in later life lends no support to the suggestion that it is the last stage of pericellular cirrhosis. Although syphilitic pericellular cirrhosis does not naturally pass into ordinary cirrhosis, it is quite possible that a liver which is recovering from this condition may, since its resistance presumably is diminished, be more susceptible to causes which make for ordinary cirrhosis. This supposition would explain the occasional occurrence of cirrhosis of the ordinary type in a child or young person the subject of congenital syphilis. This cirrhosis in a case of congenital syphilis I have recently seen, and the above explanation seems to me not improbable.

Pericellular cirrhosis is, then, an indication of the severity of the infection, and though it must interfere with the functions of the liver, and so with nutrition, we cannot disassociate its effects from those of the primary disease.

The prognosis is fairly good provided that treatment is directed to the cure of the primary disease, *viz.* mercurial inunction or hydrarg. c. cretâ gr. ss—j. t. d., and strict attention is paid to diet.

CIRRHOSIS OF THE LIVER IN RICKETS.

In children with rickets the liver and spleen are frequently palpable below the ribs, and said to be enlarged. In some of these cases the enlargement is apparent rather than real, and due to rickety deformity of the chest walls. While, again, not only is the liver normally larger in young children, but it projects below the costal arch, and hence, if judged by adult standards, would be considered to be morbidly enlarged. In infants the liver is as 1 to 20 of the weight of the whole body, while in adults the proportion is as 1 to 40.

But while bearing these cautions in mind, there are cases where the enlargement cannot be

explained away thus, and where the naked-eye and microscopic appearances show changes from the normal. These lesions are, however, less common in the liver than in the spleen or lymphatic glands. Sir W. Jenner described naked-eye changes in spleen, kidneys, lymphatic glands, and liver in rickets. He regarded it as due to infiltration with albuminoid material, but though using this term he did not mean that it was a change exactly identical with lardaceous disease. Dr. Dickinson* showed that the change was a fibrosis taking place in the portal canals. Dr. Hogben described the cirrhosis as starting from the bile ducts, and being therefore a biliary or "hypertrophic" form; the walls of the bile ducts were thickened, there was an increase in the number of small bile ducts, and a definite hyperplasia of the interlobular connective tissue which tends to surround the individual lobules—a monolobular type of cirrhosis. The cause of the biliary cirrhosis occurring in rickets, Dr. Hogben suggests, lies in the gastro-intestinal catarrh which is so commonly present. This seems highly probable, since a catarrhal condition of the larger ducts would favour the passage up into the liver of micro-organisms from the duodenum. But that catarrhal inflammation of the larger ducts does not set up obstruction to the passage of bile, and the retention of bile in the liver to biliary cirrhosis is shown by the absence of jaundice, which, if this were the cause, would be present, as we see in ordinary catarrhal jaundice.

Dr. Dickinson described the change as a fibrosis in the portal canals. The occurrence of ordinary or "venous" cirrhosis could be easily explained on the view that in rickets gastro-intestinal catarrh gave rise to the absorption of poisonous products from the intestine, which when carried to the liver by the portal vein set up inflammatory changes like those met with in ordinary alcoholic cirrhosis. In this change (rickety liver) the liver cells may show accidental fatty infiltration.

The liver is uniformly enlarged, smooth, firmer than in health, and does not give rise to ascites or to jaundice. The enlargement of the organs may pass away, and the condition appears to be a temporary one, and not to be the precursor or first stage of ordinary cirrhosis.

* 'Med.-Chir. Transactions,' 1869.

† 'Birmingham Med. Review,' vol. xxiv, p. 65, 1888.

HEPATIC CIRRHOSIS IN CHILDREN.

As in adult life so also in childhood the liver may become cirrhoued. The fibrosed organ may either be smaller than in health—the atrophic hobnailed liver—or, and this is often seen in children, it may be larger than in health. This increase in bulk may be due (1) to rapid proliferation of the fibrous tissues of the portal spaces, (2) to concomitant fatty change, or (3) to the occurrence of a special form of inflammatory change starting in and around the small bile ducts—Hanot's hypertrophic cirrhosis with chronic jaundice. In contradiction to atrophic cirrhosis any large cirrhotic liver is often spoken of as hypertrophic cirrhosis. This is unfortunate inasmuch as it is ambiguous, and may imply either the special variety of biliary cirrhosis described by Hanot, or merely a large cirrhotic liver. It would, no doubt, be better to restrict the term hypertrophic cirrhosis to the first of these two conditions,—the characteristics of which are chronic jaundice lasting for years, a tender uniformly enlarged smooth liver, green in colour, enlarged spleen, no ascites, and a haemorrhagic tendency; microscopically there is monolobular cirrhosis starting around the bile ducts, multiplication of the bile canaliculi, which contain plugs of inspissated bile, minute calculi, and preservation of the liver cells in a functional state for a long time, thus contrasting with ordinary or common cirrhosis, where they rapidly degenerate.

In children examples of Hanot's hypertrophic cirrhosis with chronic jaundice occur, and sometimes cases are met with which, conforming in most respects to that type, differ from it in presenting in addition ascites, the latter complication often only supervening towards the end.

We may, then, divide cirrhosis into ordinary cirrhosis and hypertrophic cirrhosis with chronic jaundice.

In ordinary cirrhosis, as already mentioned, the liver may be either small and atrophic or enlarged, and cases have been recorded where a large liver has been watched and seen gradually to diminish in size. It was, indeed, formerly taught that this was the ordinary sequence of events in adults, but recently doubt has been thrown on this idea that the liver of atrophic cirrhosis is the last stage of a liver which when the morbid process first began was enlarged. Micro-

scopically the cirrhosis is usually of the ordinary multilobular type, though sometimes it is rather monolobular without there being any other sign of biliary cirrhosis. From the cases I have collected it appears that in children cirrhosis is less frequently associated with tubercle elsewhere in the body than is the case in adults, where the two processes are often found together, and are probably both the result of a common cause—alcoholic excess. The microscopic appearances of hypertrophic cirrhosis with chronic jaundice have already been mentioned.

Congenital cystic disease of the liver, as I have observed it, is, from the pathological point of view, a beautiful example of biliary cirrhosis; the bile ducts become dilated, and from pericholangitis fibrosis follows the cystic dilatation, the liver cells remaining in a very fair state of nutrition. Pye-Smith * has described vacuoles in the liver cells, an appearance which, however, may well be explained as the result of invagination of the liver cells over dilated bile capillaries, which, when cut in section, give the appearance of vacuoles. Clinically, however, congenital cystic disease gives rise to no jaundice, or, in fact, to any symptoms pointing to hepatic disorder.

CAUSES OF CIRRHOSIS.

The observation of cirrhosis in children has had much to do with broadening our views as to its aetiology in general. The occurrence of a typical gin-drinker's liver in a child who has never tasted alcohol in any form renders the ordinary explanation of cirrhosis untenable for all cases.

Many examples of atrophic cirrhosis in children have no doubt been traced to the influence of alcohol, and in one case where no ordinary alcoholic drink had been taken, vinegar† had entered largely into the diet. It is usually assumed that alcohol itself acts as a direct irritant to the connective tissues around the portal vein, setting up proliferation, while at the same time it induces degeneration in the more sensitive liver cells, and some experiments on animals support this; other experiments, however, are negative, and Krawkow‡ has suggested that the rôle of alcohol in producing

* 'Trans. Path. Soc.' vol. xxxii, 1881.

† Jollye, 'Brit. Med. Journ.', 1892, vol. i, p. 858.

‡ 'Archiv de Méd. Expér. et de Anat. Path.', No. 2, 1896.

cirrhosis is that it sets up gastro-intestinal catarrh, which leads to the absorption of poisonous bodies from the intestine, which in their turn give rise to the changes of cirrhosis in the liver. Experimentally, the introduction of certain micro-organisms in large quantities into the alimentary canal have been found to give rise eventually to hepatic cirrhosis, presumably from absorption of bacterial products into the portal blood, while injections of filtered cultures of bacterial activity into the general circulation have produced the same effect.

So it appears that toxic materials, whether carried by the portal vein or by the hepatic artery, may give rise to interstitial hepatitis.

It is noteworthy that in the rare instances where two children in the same family have died of cirrhosis, that the diet they indulged in was particularly stimulating. It is possible that cirrhosis may follow faulty digestion and the absorption of toxic bodies, but though dyspepsia and cirrhosis often occur together, they appear more to be due to the same cause, rather than that dyspepsia engenders cirrhosis. In fact, it is generally assumed that when dyspepsia precedes cirrhosis it is a manifestation of the hepatic change, and not *vice versa*.

Cirrhosis sometimes follows at a considerable interval the incidence of one of the infectious fevers; thus measles or scarlet fever may, apparently by producing a toxic condition of the blood, set up changes in the liver analogous to those met with in scarlatinal nephritis. Klein* long ago described acute interstitial hepatitis occurring in scarlet fever; this condition when it occurs probably is transient, and undergoes resolution in the majority of cases, and from the frequency of scarlet fever and measles, and the comparative infrequency of cirrhosis in children, this must usually be the case. But it is reasonable to believe that under conditions, the nature of which we at present know nothing, the infectious fevers may be causal agents in the production of cirrhosis of the liver in children. In obscure conditions like splenic anaemia, where some toxic body is perhaps the cause of the symptoms, some cirrhosis is often present. In pernicious anaemia, however, which is also probably a toxic condition, cirrhosis does not occur. Long-continued chronic venous congestion of the liver—

nutmeg liver—is sometimes described as being a cause of cirrhosis; this sequence is rare, but I have seen one case in a girl in whom long-standing mitral disease gave rise to cirrhosis, though microscopically of slight degree. It is possible that alcohol given medicinally may be a causal factor; Hanot suggests that cirrhosis in *morbus cordis* is due to auto-intoxication set up by absorption of irritating bodies from the intestinal tract, which in such cases is frequently the subject of catarrhal inflammation. On the other hand, slight apparent cirrhosis might be of the nature of fibrous substitution, the atrophy of the liver cells showing up the fibrous tissue, which has also to some extent increased in amount. On the whole, chronic venous congestion is so rare and doubtful a cause of cirrhosis as to be hardly worth taking into account.

So far, then, we have seen that cirrhosis may be due to poisons absorbed by the portal vein, or to poisons reaching the liver by means of the general circulation, *i.e.* the hepatic artery.

There is another approach by which irritants may reach the liver, viz. by the bile ducts. Inflammation or the causes of inflammation may spread up from the duodenum, giving rise to cholangitis and pericholangitis in the same way that tracheitis may spread and produce bronchitis and broncho-pneumonia. Not only may the process spread upwards from the larger ducts, but it may start in the smaller ducts and extend downwards. Hunter found that in experimental poisoning with toluylendiamine the smallest bile ducts were inflamed from that drug being excreted into them.

Here, then, is a plausible explanation of biliary cirrhosis or Hanot's hypertrophic cirrhosis with chronic jaundice, *i.e.* that it is due to a poison excreted into the bile canaliculi and minute ducts which there sets up cholangitis and inflammation around. The enlarged spleen, occasional fever, hemorrhagic tendency, fit in with this view; there is a toxic condition of the blood present. There is a remarkable form of biliary cirrhosis endemic in Calcutta among the children of the coolie class, which Gibbons,* who described it in 1891, believes is due to poisons generated in the intestinal tract as the result of improper food. It

* 'Path. Soc. Trans.,' vol. xxviii, p. 439.

* Scientific Memoirs by Medical Officers of the Army of India, Part vi.

is extremely fatal; 95 per cent. of those who suffer from it die before the end of their second year.

Symptoms.—The clinical picture may be like that in an adult—emaciation, dry skin, ascites, jaundice, diarrhoea, and haemorrhages.

Jaundice appears from the recorded cases to be somewhat commoner than in adults. Epistaxis, haematemesis, and cutaneous haemorrhages are often present, and point to a high degree of disorganization of the liver tissue, hepatic inadequacy, which allows toxic bodies, which should under ordinary conditions have been arrested, rendered innocuous, or destroyed by the liver, to reach the general circulation. Headache and vomiting may in like manner be explained as being due to a biliary toxæmia analogous to uræmia. In some cases the appetite, which is usually very poor, may be ravenous or capricious. Fever may be present.

In rare instances the symptoms are entirely nervous, and cirrhosis, though practically the only lesion found post mortem, is never suspected during life. Dr. Ormerod ('St. Bartholomew's Hospital Reports,' vol. xxvi, p. 57, 1890) has described this group of cases: there is progressive paralysis and loss of mental power going on to idiocy, fever and marked emaciation. Post mortem, in addition to cirrhosis, small patches of softening on the lenticular nuclei, the significance of which were uncertain, were found. In less marked cases coma, delirium, and screaming may come on shortly before the end, and may be the result of considerable destruction of liver tissue taking place, perhaps to a process akin to acute atrophy supervening (icterus gravis).

It would be interesting to know whether there is any relation between the absence of ascites and the presence or the tendency to the development of nervous symptoms—whether ascites after all serves a useful end by diverting the poison from the nervous system, the peritoneum excreting the toxic substances and so trying to protect the nobler nervous system. In some cases where the nervous symptoms have been prominent, ascites has been conspicuously absent. In other cases after tapping, non-accumulation of the peritoneal fluid coincides with the development of nervous symptoms, but, on the other hand, they may both be present together, or the cerebral phenomena may only supervene towards the end of a case in which ascites has been present through-

out. The important symptoms are ascites and jaundice. Paracentesis may be required, and when this has been performed, the end, if the case be one of cirrhosis, is not far off. In some cases paracentesis has had to be often repeated, and in one instance it was done 36 times; here there was chronic peritonitis, and this opens up the question whether the ascites was due to the chronic peritonitis or *vice versa*. Hale White* has shown that in adults, cases of cirrhosis alone seldom last long enough to require tapping more than once, and that if this is necessary there is probably chronic peritonitis as well.

These are the symptoms commonly present in ordinary cirrhosis, though the nervous phenomena, at any rate to such a degree as described by Dr. Ormerod, are rare. Less commonly an enlarged tender liver, enlarged spleen, fever, haemorrhage, and obstinate jaundice, without ascites, make up the morbid entity sometimes called *maladie de Hanot*, after the observer who first described the condition as "hypertrophic cirrhosis with chronic jaundice."

Diagnosis.—It may be when the symptoms are entirely nervous that cirrhosis is never suspected; on the other hand, with jaundice, ascites, and a big liver the diagnosis is easy. But when ascites alone is present there may be difficulty in distinguishing it from tubercular peritonitis: the absence of any enlarged mesenteric glands, the tense not doughy feel of the abdomen, and the occurrence of haemorrhage are in favour of cirrhosis; while the tuberculous aspect, enlarged glands in the abdomen or elsewhere, and turbid fluid drawn off by paracentesis point to tubercular peritonitis. Abdominal tuberculosis is perhaps the disease with which cirrhosis is most likely to be confused; but when the course is somewhat acute and accompanied by fever, the suspicion of generalized tuberculosis or of typhoid fever may arise. Dr. C. West speaks of cases with fever, emaciation, dry harsh skin, and ascites, which suggest grave organic disease, but in which a cure resulted from quinine, and were therefore regarded as malarial. Such cases are difficult to diagnose, especially as malaria is now rare, but the observation indicates a trial of quinine on the chance of a successful result.

The *prognosis* is extremely bad, and when

* 'Guy's Hospital Reports,' 1892.

symptoms have sufficiently developed to warrant a diagnosis it is probable that the issue is invariably fatal. Targett ('Path. Trans.', vol. xl, p. 136), however, has described a typical hob-nailed liver in a child aged 8, who, when presumably in perfect health, was killed by an accident; and from this we may conclude that it is possible that in children as well as in adults cirrhosis of the liver may become arrested, and though not cured, sufficient compensation may take place to restore the balance of health. Statistics show that in adults the condition of arrested cirrhosis is associated with a large liver.

Treatment.—In the first instance the avoidance of any stimulating food or drink is absolutely necessary, and a simple diet, of which milk is the staple, should be adopted. Fatty foods such as cream, butter, cod-liver oil, should, if they do not upset digestion, be given, and theoretically, since they pass through the thoracic duct, should be more easily absorbed than protein and saccharine food, which has to traverse the obstructed portal circulation.

If ascites is present the patient should be in bed, but otherwise fresh air and a moderate degree of exercise are desirable. There is no drug which can be relied upon to in any way absorb or remove the newly-formed tissue in the liver, but mercury in the form of hydrarg c. cret, gr. ss—j. t. d., may be tried, and the syrup of the iodide of iron, or some bitter, such as gentian or calumba, may be given, while the bowels may with advantage be kept open with Carlsbad salts or some other saline. An attempt may be made to diminish ascites by salines and diuretics, of which digitalis, squills, and copaiba may be given a trial, and the amount of fluid taken should be somewhat restricted. But, as already mentioned, ascites is usually a sign that death is not far distant, and on the whole paracentesis, inasmuch as it gives relief, should not be postponed after the abdomen has become much distended.

DR. CANTRELL firmly believes in the utility of treating both *furuncles* and *carbuncles*, if seen early, with ichthyol, and what would seem impossible is that he has seen both of these conditions aborted when treated early.

Philadelphia Polyclinic.

A STUDY OF APPENDICITIS.

By PROFESSOR DIEULAFOY.

(*Bulletin de l'Académie de Médecine.*)

DURING the last few years inflammation of the cæcal appendix has taken, both in medicine and in surgery, an importance of the first order. Acute generalized peritonitis, subacute peritoneal septicæmia, partial and encysted peritonitis, secondary peritoneal abscesses, circumscribed hepatic abscess, as accidents or as complications, always serious, often dangerous, are due to lesions and infections originating in the appendix, which one is accustomed to include under the designation of "appendicitis." *

It must, from the first, be well understood that it is to appendicitis, and to this alone, that we should refer all the symptoms, all the accidents hitherto imperfectly recognised and ill-interpreted, which were previously attributed to typhlitis and perityphlitis.

Our knowledge of appendicitis is of recent date. Until 1888 typhlitis reigned supreme.† This faecal typhlitis was said to be due to an obstruction of the cæcum by faecal accumulation. Such obstruction, block, or stoppage of the bowel, as it is called, set up inflammation of the walls of the cæcum. Here was typhlitis with its train of symptoms—constipation, pain, swelling, fulness of the right iliac fossa, and in the more acute cases fever, nausea, and vomiting.

This so-called typhlitis usually ended in resolution; but it might lead to ulceration or to perforation, and so excite perityphlitis, peritonitis, or iliac phlegmon. It was further said there would be peritonitis if the lesion were on the anterior wall of the cæcum, and cellulitis if on the posterior wall, which was wrongly supposed to be uncovered by peritoneum.

Such were, in a few words, the notions which for a long time received acceptance. However,

* The opinions which I put forward in this communication are opinions which I have expressed and fully developed in the thirteen lectures given upon Appendicitis during November and December, 1895, in the Faculty of Medicine.

† Richard. "Typhlite, Perityphlite, et Appendicite," *Gazette des Hôpitaux*, 1891, No. 17, p. 145.

all this, we may at once say boldly, was a tissue of errors, for in the sense just stated stercoral typhlitis does not exist. To begin with, it is not true that the posterior surface of the cæcum is uncovered by peritoneum. In 1887 * Tuffier confirmed the opinion, too long left unnoticed, that never—neither in the adult nor in the foetus—is the cæcum in direct contact with the cellular tissue of the iliac fossa; the cæcum is surrounded by the peritoneum, and “the hand can pass round it as it can pass round the apex of the heart in the pericardium.”

Moreover, about the same time the American surgeons were led to open the abdomen without delay for the accidents previously imputed to typhlitis, recognising that these accidents had their origin not in the cæcum, but in the cæcal appendix. The same observation was made hundreds of times by the surgeons of all countries—in England, Switzerland, Germany, and France—with the result that for the old and erroneous description of typhlitis was gradually substituted the precise and correct notion of appendicitis. These ideas, grudgingly admitted by some, were once more confirmed at the last Congress of Surgery; and in the same year, in 1895, in the course of the brilliant discussion of the Société de Chirurgie, Routier † definitely stated that in the large number of cases under his own observation it was always the appendix which was the starting-point of the accidents, and not the cæcum. I have had occasion to witness a large number of operations for appendicitis, and I am absolutely convinced that it is always to the appendix, and never to the cæcum, that we must refer the symptoms and the accidents of all kinds which were formerly attributed to typhlitis.

Are we, then, to say that the word “typhlitis” must be absolutely removed from our nosological list? No; but it must be properly understood. The ulcerations of enteric fever (typhoid), and tubercular ulcerations of the intestine often reach the cæcum, but these specific lesions are outside the question which we are considering. Tuberculosis may be seated in the cæcum as a primary

* Tuffier. “Étude sur le cæcum et ses hernies,” Archives Générales de Médecine, 1887, vol. i, p. 641.

† Routier. “Discussion on Appendicitis,” ‘Bulletins et Mémoires de la Société de Chirurgie,’ 1895, t. xxi, p. 531.

affection—slow, chronic, within reach of operation, and in that way curable; but this primary and chronic tubercular typhlitis, which so closely simulates cancer, has nothing in common with the typhlitis which forms the subject of this discourse.

A perforation of an uncertain nature may possibly affect the cæcum as it may the ileum;* but once more, all these cases, set aside because they have their seat in the cæcum, have nothing in common with the question in dispute, and it remains well established that it is to appendicitis that we must refer all that had been accredited to typhlitis.

But, after all, it will be said there are undoubtedly cases where one has found the lesions of typhlitis, ulceration, gangrene, suppuration. Yes; but when we look closely into these we shall see that these lesions of typhlitis are themselves consecutive to the lesions of appendicitis; the appendix primarily affected is, in some positions, close beside the posterior surface of the cæcum. Such was the case of Gambetta.†

These views being well established, let us study the pathogenesis of appendicitis.

Pathogenesis.—The theories concerning the pathogenesis of appendicitis are derived from the theories which were put forward to explain the pathogenesis of what was supposed to be typhlitis. Sometimes constipation, sometimes diarrhoea was accused, without knowing exactly which of these two extreme conditions was the most to blame. The cause was sought in foreign bodies, fragments of bone, the scales of fish, bits of egg-shell, pins, needles, plum stones, the pips of fruit, and who knows what.

I will say at once that foreign bodies more or less voluminous, such as fragments of bone, the stones of plums, dates, cherries, etc., naturally capable of producing injury in the cæcum, are absolutely incapable, from their size, of entering the narrow orifice of the canal of the appendix. At most we may inculpate the small foreign bodies, such as the pips of grapes; and yet, since the question is now better understood, since the concretions

* Letulle has published an interesting case of perforation of the ileum of unknown nature. Letulle—“Des perforations aiguës de l'intestin grêle,” ‘Presse Médicale,’ 1885, p. 137.

† Lannelongue, Cornil-Blessure. “Maladie de Gambetta; Observation, Autopsie,” ‘Gazette hebdomadaire de Médecine et de Chirurgie,’ 1883, No. 3, p. 33.

of the appendix have been better studied, one sees that these concretions, which may assume somewhat the form of grains of coffee, date stones, grains of wheat, or beans, are nothing more than calculous concretions of which the formation and structure are now well known.

It is the presence of these calculi in the canal of the appendix which has given to this variety of appendicitis the term "calculous appendicitis."

When a surgeon is called on to perform an operation for the accidents of appendicitis or of appendicular peritonitis, it is in the appendix that one finds the calculus if an infecting process has not destroyed or perforated the appendix; but if the appendix is perforated, necrosed, or gangrenous, the calculus may have passed into the peritoneum, and is found either at the time of operation or during the next few days in the dressings.*

These calculi of the appendix are exceptionally well discussed in the interesting thesis of Rochaz,† presented last year at Lausanne. In this work will be found the description and the reproduction in plates of sixty-five calculi or concretions of the appendix, forming part of the observations of Roux (of Lausanne).

Calculi of the appendix have the most variable size, and one is surprised to see some of them which have attained to the dimensions of a plum stone, and beyond. They are, so to speak, never round; they are more or less elongated, fusiform, cylindrical. Usually a single calculus only is found, but one may see even three or four; they then show several facets. The usual colour of these calculi is brownish; their consistency is most variable. Sometimes they are soft and friable without cohesion; sometimes dense, firm, or of stony hardness, with all intermediate degrees. These calculi are formed of several elements, which enter into their composition in variable proportions. Berlioz has made for me a minute analysis of several calculi of the appendix which I entrusted to him, and the result of his researches accords absolutely with the results obtained by other chemists quoted by Rochaz in his thesis

(Volz, Bulter, Pelet). One finds in these calculi an organic stercolar matter, brownish, in great part soluble in ether, and some mineral salts, chiefly calcareous, phosphates, and carbonates of lime; at times salts of magnesia, traces of chlorides, sulphates, and very rarely cholesterine (Walt). These different elements, organic and mineral, are cemented together by the mucus which is abundantly secreted by the glands of the appendix.

One understands then why the concretions of the appendix are sometimes soft and friable, sometimes hard and as though calcified. If the concretion is chiefly rich in organic matters it is soft, or at least it easily softens when infection of the appendix is set up. If the concretion is chiefly rich in mineral elements and in lime salts, it may take a lithic consistency like a true calculus. It is easy

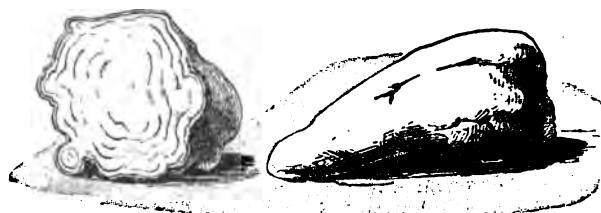


FIG. 1.—Stratified calculi from the appendix.

to trace the method of formation and evolution of these calculous concretions of the appendix: a section demonstrates that more frequently these concretions are *stratified*. (Fig. 1.) Around one or more cores are formed successive excentric layers, more or less irregular, of which the stratification proves to demonstration that the calculi of the appendix develop and enlarge slowly and progressively in the canal of the appendix by the addition of organic and mineral layers. This process is very evident in the appendicular calculi which I gave to Berlioz. The section of one good-sized calculus of these showed stratification as well marked as the stratified sections represented in Rochaz' thesis.

It was in studying the process which I have described, in comparing the slow and progressive formation of calculi of the appendix with the entirely analogous formation of biliary calculi, that the idea suggested itself to me to propose for the formation of calculi of the appendix the term *lithiasis appendiculaire*, lithiasis of which the connection seems to me quite close, as we shall see later, with urinary and with biliary lithiasis.

* Loison. "Contribution à l'étude pathogénique et thérapeutique de l'appendicite ulcéro-perforante," 'Revue de Chirurgie,' t. xv, 1895, pp. 1—25.

† Rochaz. 'Contribution à l'étude des calculs appendiculaires,' Thèse, Lausanne, 1895.

What becomes, then, of the old theory of Talamon, a theory according to which the calculi get into the appendix, having been originally formed in the cæcum? According to the statements of Talamon, it is in the cæcum that the scybalæ are rolled and pressed, they are there rounded like pellets under the finger,* and after this *quasi*-pill-making process they are sent from the cæcum into the appendix. This theory has been demolished by the arguments of Rochaz, which I proceed to quote textually. "It is on the perfectly spherical form of the calculi that Talamon relies to found his theory; but this form, so perfectly spherical, we have only met with three times in our sixty-five cases, and the form which we met with almost always, and which is the rule, is just that elongated cylindrical shape which Talamon has never met with, the form admitted by the authorities. How are we to explain by the pellets of Talamon the long stercoral sausages filling the appendix from one end to the other? How could these large concretions pass fully formed through an opening which at the most is no more than five millimetres in diameter, and which is in addition more or less closed by a valve? On the contrary, the disposition of the calculus in concentric layers indicates a slow formation, which one can only explain as occurring in a recess separated from the intestinal tube, and not in the cæcum, where these calculi would be carried away after a short time by the flow of the intestinal contents."†

The question is, therefore, settled. To the excellent arguments advanced by Rochaz against the theory of Talamon I have nothing to add; but if Talamon thus holds to his theory, which will not stand serious investigation, it is that he needs it to support another theory, equally erroneous, that of colic of the appendix, which we shall come across presently.

For the moment it is an established fact; this is, that the form of appendicitis termed calculous is associated with a process of lithiasis in the appendix, comparable in its genesis with biliary and with urinary lithiasis.

But appendicitis is not always of calculous origin; this may be absent. There are cases, and

they are sufficiently numerous, where appendicitis may arise with all its most serious consequences, general peritonitis and encysted (localised) peritoneal abscess, without one being able to find the least calculous concretion in the interior of the canal of the appendix.

We have to do in such cases with a local infection, which the American surgeons have called, for want of a better term, Catarrhal appendicitis. That this denomination has been criticised matters little; it establishes in every case a well-marked distinction between the appendicitis which is due to calculus and that which is not.

Several authors refuse to admit of an appendicitis not dependent on calculus. If we do not always find the calculus in the appendix, it is, they say, that the calculus may have fallen into the peritoneal cavity through a necrosed or perforated appendix; or possibly again, they say, the calculus of the appendix may have passed into the cæcum, which will explain its absence from the appendix. These arguments are valueless. That the calculus may escape into the peritoneal cavity through a perforation of the appendix is certainly true, witness the cases quoted by Loison, where two large calculi from the appendix which had fallen into the peritoneal cavity were recovered several days after operation, between the lips of the skin incision and on the linen of the dressings. But what value can such arguments have when we are dealing with appendicitis without perforation? As to the other argument, that which consists in the supposition that a calculus of the appendix can get back into the cæcum, it will not stand discussion. How can we admit, in fact, that a calculus, the dimensions of which are three or four times as great as those of the canal of the appendix, could pass along this canal, which is normally so narrow, and which is still more narrowed and even obliterated in the case of appendicitis? We must, then, submit to the evidence: there is a calculous appendicitis and a non-calculous appendicitis. Whether one calls this latter catarrhal or infective, what you will, it is no less true that this form may be as severe as the other, it may equally lead to peritonitis and to all the accidents, with or without perforation of the appendix. These conditions are recorded by a large number of observers. I have many times confirmed them.

* Talamon. 'Appendicite et perityphlite,' p. 45 (Collection, Charcot-Debore).

† Rochaz. Loc. cit., p. 19.

It remains now to elucidate the pathogenesis of appendicitis and the pathogenesis of the terrible accidents which may result there from it. This is the place to refer to experimental researches and to bacteriological investigation.

In 1889 Clado presented to the Congress of Surgery a remarkable clinical and experimental work on hernial infection. He had observed that the micro-organisms of the strangulated portion of intestine penetrate into the hernial sac without there being any perforation of the intestine, and this on the day after the strangulation. He followed the migration of the microbes through the non-perforated coats of the intestine; he found that the peritoneal cavity might be invaded in its turn; he, in short, demonstrated the possibility of infective accidents by generalization of the microbial agents.

Bennecken, Oker-Blom, and many others have repeated these experiments, and have shown that the *Bacillus coli* imprisoned in a hernial loop of the intestine, strangulated, and invaginated, may pass into the peritoneal cavity through the coats of the intestine or by the lymphatic tract, and thus set up a peritoneal infection.

But the most remarkable work undertaken on this subject is that of Klecki.* With all antiseptic precautions, Klecki strangulated a loop of intestine in a dog by means of an india-rubber ring. After twenty-four or forty-eight hours the dog died or was killed, the strangulated loop of intestine was nowhere perforated, and yet it had given rise to a peritonitis.

I cannot follow Klecki through all the details of his experiments, but the following are the principal results which spring from them.

In the loop of intestine experimentally converted into a closed cavity, a large brood of the microbes habitually found in the intestine is produced, with a great exaltation

of their virulence. These microbes, in their new pathological condition, are able to pass through the perforated intestinal wall, and determine a consecutive peritonitis.

The virulence of the microbes is more violent in the intestinal loop transformed into a closed cavity than it is in the peritoneum.

It is therefore not in the peritoneal cavity that we must look for the key of the question. The *Bacillus coli* and other microbes which form the poly-infection reach the peritoneum through the pathological loop in which they have already undergone biological changes which exalt their own virulence and the virulence of their toxines.

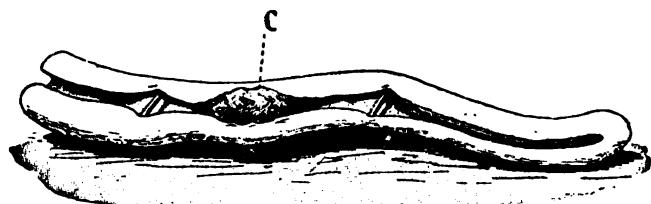


FIG. 2.—Obliteration of the appendix by a calculus, C.



FIG. 3.—Obliteration of the appendix by swelling of its walls at the point R.

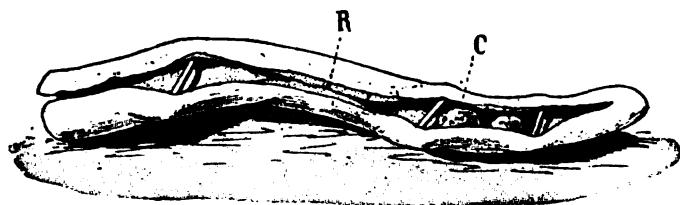


FIG. 4.—Obliteration of appendix by swelling of its walls at point R. In the closed cavity there are two calculi freely moveable.

Let us now apply these experimental results to the pathogenesis of appendicitis.

The appendix itself, being a diverticulum of the intestine, may become transformed into a closed cavity by the various methods which I have reviewed, and which I have had repro-

* De Klecki. "Recherches sur la pathogénie de la péritonite d'origine intestinale: étude sur la virulence de coli-bacille," *Annales de l'Institut Pasteur*, t. ix, pp. 710—736.

duced by Bonnier in the figures annexed to this work.

Sometimes it is a calculus of the appendix, which, in its slow and progressive growth, at length obliterates the canal of the appendix in some part of its course. (Fig. 2.)

Sometimes it is under the influence of a non-calculous inflammation by the swelling of the mucous membrane and of the coats that the canal of the appendix is obliterated at its orifice, or in some other part of its course, exactly as the Eustachian tube is obliterated in the case of otitis. (Fig. 3.)

Frequently, also, these two processes are found conjoined; a calculus may have favoured a local infection, and the subsequent tumefaction of the walls completes what the calculus had commenced. (Fig. 4.)

There are also some cases in which a fibrous stricture forms slowly in some portion of the canal of the appendix. Such was the case published by Achard;* the appendicitis had led to perforation of the appendix, to a centre of perityphlitis, to abscesses of the liver, and yet there was no calculus: the starting-point of infection in the appendix was consecutive to the obliteration of the orifice of the canal by a tissue of fibrous appearance. Rendu has published a case of purulent appendicitis set up not by a calculus, of which there was no trace, but "by a sort of fibrous partition which separated the diseased appendix from the healthy cæcum;" it was easily seen that the communication between the cæcum and the appendix was completely obliterated.†

From all this it follows that the canal of the appendix may be obliterated, either at its cæcal orifice or at some part of its course, by different processes. This obliteration may be due to a calculus progressively increasing in size: it may be due to a swelling of the walls resulting from a local infective process; it may be due to a process of fibrosis. Frequently also, as I have shown, several of these causes may be found united: the calculus of the

appendix and the inflammatory swelling of the walls each contributes its share to the partial obstruction of the canal of the appendix. This obstruction is temporary or permanent according to the nature of the obliterating process. But whatever the cause of the obstruction, whether this obstruction be due to a calculus, to a swelling of the walls, or to a fibroid narrowing, or to several of these causes together, the essential fact, the fact which dominates the whole history of appendicitis, is that the *portion of the canal of the appendix which adjoined the obstruction is converted into a closed cavity*. Further, the microbes of the appendix which in the normal condition were inoffensive, as are all the microbes of the free intestine, these microbes when imprisoned have their virulence increased, as in the experiments of Klecki; they then become a centre of "polyinfection," in which the *Bacillus coli* and the *streptococcus* are the principal agents, as I have shown in conjunction with my "Interne" Kahn, and one may say that from this moment appendicitis is established.

Yes, appendicitis is established, as one may say, from the instant when the cavity of the appendix is transformed into a closed cavity, with or without calculus, it matters little; the patient is in danger of multiple accidents, sometimes serious, too often dangerous, as will be explained immediately.

Such is the theory of appendicitis from a closed cavity which I think I have been able to form, thanks to the beautiful experiments of Klecki. This theory in addition receives fresh confirmation from the interesting experiments of Roger and Josué.*

These authors have ligatured antiseptically the appendix in the rabbit, taking care to secure the vessels. They killed the animals some time afterwards, and they showed that the part beneath the ligature was transformed into a purulent cavity. They concluded from this, and with good reason, that it sufficed to imprison the microbes of the appendix, "to transform the inoffensive microbes of the intestine into pathogenic agents."†

* Achard. "Infection du foie compliquant l'appendicite; pathogénie des abies aréolaires," 'Bulletins et Mémoires de la Société Médicale des Hôpitaux,' 1894, p. 793.

† Rendu. "Discussion sur l'appendicite expérimental," 'Bulletins et Mémoires de la Société Médicale des Hôpitaux.' 1896, No. 4, p. 81.

* Roger and Josué. "Experimental Appendicitis," 'Bulletins et Mémoires de la Société Médicale des Hôpitaux,' 1896, No. 4, p. 79.

† The new theory of appendicitis, which I maintain differs on many sides from the ideas put forward by Talamon. According to Talamon, the calculus "engages suddenly in the appendix by a violent contraction of the

Let us now turn to the pathogenesis of appendicitis from another point of view, I refer to *heredity*. Roux (of Lausanne) has already maintained with justice that appendicitis is frequently hereditary. For my part, experience teaches me that one often sees appendicitis in several members of the same family, that it shows itself in collaterals or in descendants. In observing more closely it has appeared to me that this heredity shows itself chiefly in families affected with gout, arthritism, urinary and biliary gravel, so much so that I have proposed to make over appendicular lithiasis to the patrimony of arthritism and gout. Appendicular lithiasis produces its effect in the canal of the appendix, as biliary lithiasis in the gall-bladder, as urinary lithiasis in the kidney; and it seems evident to me that this new lithiasis should itself also take its place amongst the faults of nutrition studied by Bouchard. Arthritism, obesity, biliary lithiasis, gout, diabetes, lithiasis of the kidney and of the appendix, are various possible manifestations, hereditary or acquired, of the same diathesis. Here are some examples.

One of my best pupils is occasionally attacked

cæcum, penetrates it, and becomes wedged in the upper portion of the narrow canal." Rochaz, as we have seen above, has shown the true value of this theory. According to Talamon, one of the consequences of a calculus so impacted is "the compression of the walls of the appendix and the hindrance of the circulation in the vessels contained in the walls;" then "the microbes, inoffensive, and impotent against the healthy elements, triumph without difficulty over these elements when deprived of the nourishing liquid of the blood." Roger and Josué, we have seen, have shown what this hypothesis is worth; they took great care in their experiments to protect the vessels of the appendix, but the focus of inflammation in the appendix was none the less produced. Thus their experiments, like the clinical facts of appendicitis without calculus, condemn the theory of Talamon. However, Talamon hit upon a happy expression—"the closed vase;" but this expression was stillborn, lost as it was in the midst of erroneous theories. According to Talamon again, the painful symptoms at the onset of appendicitis are nothing more than "appendicular colic," due to the passage of a cæcal calculus into the canal of the appendix, as hepatic colic is due to the passage of calculi in the biliary ducts. We shall see in a moment that this theory is not only erroneous, it is unfortunately dangerous, for it inspires a false security, it confuses the indications which should lead to a timely operation and to safety. Dr. Talamon has thus been badly inspired in his loud protestations accusing others of appropriating his ideas. His ideas form a tissue of errors. No one would ever dream of their "appropriation."

with nephritic colic; his father was gouty, his brother died of appendicitis.

We lost a few years ago one of our colleagues from diabetes; his daughter had died from appendicitis. I saw in 1891, in the Rue du Général-Foy, a child of twelve years who died of appendicitis; one of his uncles had been carried off by the same complaint, and his father has had several attacks.

One of the appendices, which had served me for my work, had been taken by Routier from a young girl of a family in which three persons had been attacked with affections of the appendix.

I have been for several years the medical adviser of a family in which gout, obesity, and diabetes prevail. I am aware of three cases of appendicitis in this family, of which one in a child of ten years was fatal.

Berger operated a few years ago on a man of middle age affected with appendicitis; two years later the son of this gentleman was operated on for appendicitis by Segond.

I know a lady who for fifteen years has been subject to rheumatism and to hepatic colic; on my advice her son has been operated on for calculous appendicitis by Routier.

I was called several months ago to a lady aged seventy-two years, who had been suddenly seized with appendicitis, which I got Routier to operate on; two months later it was the turn of her granddaughter to be operated on, also by Routier, for the same complaint.*

Since I have drawn attention to this question the observations have multiplied. My very distinguished colleague, Faisans,† has communicated to the Société Médicale des Hôpitaux six cases of family appendicitis observed by him or by his colleagues, and all these cases concern families in which two, three, or four persons of the same family have been attacked with appendicitis, too often fatal. At the same meeting Rendu communicated similar cases of very great interest.

* This young lady had been confined three weeks previously by Budin. Pregnancy seems to me to favour lithiasis of the appendix as it does biliary lithiasis; I have observed several cases of this.

† Dieulafoy. 'Cours de pathologie interne de la Faculté de Paris,' semestre d'hiver, November, 1895.

‡ Faisans. "De l'appendicite familiale," 'Bulletins et Mémoires de la Société Médicale des Hôpitaux,' 1895, No. 8, p. 228.

The Société de Chirurgie on its side has studied the subject from the same point of view (Brun,* Berger, Tuffier, Jalaguier, Quénu), so much so that we have lithiasis of the appendix, a family and hereditary malady, now occupying the place which it ought long ago to have occupied in our nosology, by the side of biliary and urinary lithiasis. I would say, indeed, that of the three lithiases it is the one which appears generally first, for it is frequent in children; it is the one which is the most dangerous, for the accidents which follow gravel in the kidney or biliary calculi are not comparable, from their gravity or from their frequency, with the accidents consecutive to lithiasis of the appendix.

However, it must not be thought that appendicitis is always associated with the arthritic diathesis. All cases of appendicitis are not calculous. True, the appendicitis due to lithiasis of the appendix is of arthritic origin and often runs in families; but we must count also with the non-calculous forms of appendicitis, offshoots of an infective process of the appendix or of the appendix and cæcum comparable with the obstruction of the bile-duct in cases of inflammation extending from the duodenum. This variety of appendicitis has nothing to do with arthritism nor with heredity.

All these points with regard to the pathogenesis of appendicitis being well established, let us pass to the description of the illness.

(*To be continued.*)

THERAPEUTICAL NOTES, &c.

Hysterical Aphonía.—Boulay recommends introducing a sound into the larynx far enough to cause a slight spasm or cough, and, removing the sound, have the patient call out the letters of the alphabet slowly and calmly until a sound can be produced, then to count up to ten, then to articulate simple words, and finally to converse. The sound may be introduced a second or third time, if necessary, in order to produce the desired effect. If this means fail, the aphonia is of a rebellious character, and recourse must be had to external

* Brun: "Appendite familiale." Routier, Jalaguier, Tuffier, Quénu, Berger: "Discussion sur l'appendite familiale: Séance de la Société de Chirurgie du 22 Janvier," 'Presse Médicale,' 1896, No. 8, p. xxvi, et No. 10, p. xlvi.

or internal electrilization of the larynx by means of the galvanic or faradic current for four or five minutes; having the patient perform vocal exercises while the current is being passed. Massage of the anterior portions of the neck, methodical traction of the tongue, rhino-pharyngeal palpation, or auto-laryngoscopy are psychical measures that may succeed, as well as compression of the ovaries. General treatment with strychnine, valerian, the bromides, hydrotherapy, or isolation must be associated with the local means employed, while lesions of the nose, pharynx, or larynx must also be looked after.—*Gaz. Hebdomadaire de Méd. et de Chir.*

Influenza.—In cases seen early, especially those presenting pleurodynia or pleuritic symptoms, Dr. S. Solis-Cohen, of Philadelphia, administers the following recipe: B. Salol, 3 gr.; terpine hydrate, 3 gr. Mix. In powder or capsule. One powder or one capsule is given every two, three, or four hours, according to the indications of the case, and this treatment is kept up for from twenty-four to thirty-six, or even forty-eight hours, according to the progress of the symptoms and of lesions, in case the pleura or lung be involved. In the latter case strychnine sulphate from $\frac{1}{160}$ to $\frac{1}{30}$ gr., according to the frequency of administration and the urgency of the symptoms, is combined with it. If cough is sufficiently troublesome to require sedatives, codeine (from $\frac{1}{24}$ gr. to $\frac{1}{16}$ gr.) is likewise added. Codeine is especially useful in cases of pleurisy, not only relieving pain, but also seeming to have a certain controlling influence upon the inflammatory process. Its value in inflammation of serous membranes is well known, and is, of course, the explanation of the latter fact.—*Philadelphia Polyclinic.*

Rectal Ulcers and the Electro-Cautery.—Beach, with great care and perspicuity, in an interesting article on this subject, states that the rational signs of rectal ulcers are: pain in loins, hips, thighs, and abdomen; morning diarrhoea (especially in cases of tubercular ulcer), and not uncommonly nervousness and frequent micturition. It need hardly be mentioned that general emaciation follows in the train of symptoms. The diarrhoeal discharges contain muco-purulent material, occasionally blood, which appears in the first portion of the stool. Patients presenting

themselves with the ordinary symptoms—diarrhoea, pain, &c.—have frequently baffled the skill of the practitioner who relied upon the internal administration of various drugs. But what once puzzled the enthusiastic therapist now stands forth in clear light, through his knowledge of pathology and approved methods, so that hope can be offered in the prognosis of many rectal lesions which were formerly hopeless. To arrest molecular death of the part is the first consideration, and cleanliness is above all to be desired. Rest is another factor of prime importance. To rest and cleanliness are added such measures as will restore the part *cito, et jucunde*. This applies to disease of the sigmoid as well. The special object of the writer is to present the claims of the electro-cautery as a remedial agent in certain cases of rectal ulcer, and to this end he reports two selected cases. The first had already been operated on twice by division of the sphincter, but without relief. On examination there was found an anal skin-tag beneath which was a marked irritable ulcer. The speculum was inserted, the cautery applied over the entire surface of the ulcer, and a furrow made over the long axis. Very little pain attended this application, and very slight soreness was felt the following day. Three such applications, made a week apart, resulted in complete recovery. The second case was one of chronic ulcer of the bowel. The electro-cautery was applied to the ulcer twice a week for a month, then once a week for a month, resulting in cure.—*Matthew's Medical Quarterly*.

The Indications for Operation in Puerperal Sepsis.—According to views published by Dr. Lewis S. McMurtry, we are given to understand that pre-existing pus trouble, uterine fibroid, or ovarian dermoid converted by the trauma of labour into activity as an infecting source, should be treated by prompt resort to abdominal section. Septic endometritis, with or without putrefactive changes in retained clots and *débris*, should be removed by cleansing, antisepsis, and drainage. Thorough intra-uterine drainage and irrigation in appropriate cases arrest the septic process. Curettage in these cases, when the septic focus is limited to the uterine cavity, is too extensively used. The granular area of Bumm may be broken through, closed sinuses and veins at the placental

site reopened—repeated chills and rising pulse and temperature marking the invasion of new areas of infection—and nature's barriers to increasing infection torn away by the indiscriminate use of the curette. Plugging up the uterine cavity is positively contra-indicated; drainage should be facilitated and not obstructed in these cases. Purulent salpingitis, ovarian abscesses, and suppurative peritonitis, by progressive steps, may extend very rapidly, and the associated peritonitis may be circumscribed or diffuse; consequently careful and deliberate judgment must be exercised before resorting to cæliotomy. The time of the operation and the extent to which the operative procedure is to be carried also require sound judgment. Diffuse septic parenchymatous metritis and purulent metritis should be promptly treated by hysterectomy. Puerperal sepsis, wherein the local symptoms are those of diffuse peritonitis without localisation of lesions, but wherein the uterus is presumably the focus of infection, is a grave condition and often justifies exploration and drainage, but hysterectomy will almost invariably prove disastrous.—*American Journal of Obstetrics*.

The Etiology and Treatment of Ozæna.—Belfanti and Della Vedova gave the results of their researches on this subject, carried out at the Institute of Serum-therapy, in Milan. They regard the disease as undoubtedly of bacterial origin, and due to a bacillus identical with that of Loeffler in form and characteristics, but differing from it in a great attenuation of virulence, causing only œdemas and moist gangrene at the site of the injections in guinea-pigs. The micro-organism is found in the exudate in ozæna, in the depth and on the surface of the diseased mucous membrane, and gives rise to a chemical change in the secretion and to atrophy of the mucous membrane and the bone.

In view of these facts, the authors decided to employ the anti-diphtheritic serum in the treatment of the disease, and they give the details of 32 cases, 16 of which were cured, 7 almost cured, 5 improved, and 4 showing slow improvement. The uncured cases are still under treatment, and the authors look for successful results in these also. The method of treatment consists in making injections of 10 cubic centimetres ($2\frac{1}{2}$ fluid drachms) of antitoxin every two days, or every day if possible, until about thirty injections had been given, the

number varying according to the age, duration of the disease, and local and general reaction of the remedy. The changes produced were: (1) a turgescence and congestion of the diseased mucous membrane, (2) disappearance of the characteristic odour, (3) appearance of fluid exudate, and (4) disappearance of green crusts. These alterations varied as to the time of their manifestation and their intensity. The complications produced during the treatment were neither severe nor dangerous, though the authors recommend suspending the injections until they disappear.

Bozzolo reported two cases treated by him with the antitoxin; the first a girl, 13 years of age, with chronic ozæna of an exceedingly fetid character. The odour entirely disappeared after the fourth injection, but on suspension of the treatment it returned after some time. Three injections were then made, and the odour again disappeared. Multiple subcutaneous haemorrhages made it necessary to abandon the treatment. Soon after the girl developed measles. At the time of report the condition of the nasal cavities was excellent. The second case was that of a woman of 43 years, in whom the bad smell disappeared after the fifth injection of serum. She was still under treatment at the time of report.

Gradenigo stated that he had treated sixteen cases of ozæna with the serum. In five of these the bacteriological diagnosis had been made by Belfanti. In all there had been an improvement, though the number of injections had not as yet been sufficient to cause complete cure. One case of purulent ozæna had been particularly benefited. He had noted a specific, elective action of the serum upon the diseased mucous membrane.

La Settimana medica.

The Pathology and Treatment of Rickets.

—The *Journal des Praticiens* describes the method which it believes should be resorted to in the treatment of the rickets of early life. In the first place it must be remembered that the disease depends upon an insufficient absorption and assimilation of lime salts and other substances adapted to the formation of healthy bone-tissue; second, we must not forget that according to one theory the bones are decreased in strength by a process of auto-intoxication which is generally associated with an artificial milk diet and with

dilatation of the stomach; third, we must remember that some cases of infectious disease produce rachitic tendencies, and that malaria and syphilis are peculiarly apt to do so. Whatever may be the theories as to the pathogenesis of rickets, the *Journal des Praticiens* points out that the treatment is fairly clear. In the first place, the greatest possible attention must be paid to the diet during the early months of life. Care should be taken to see that the food contains the normal proportions of bone salts, and as much fresh air as possible should be obtained by the patient. In addition to this hygienic treatment it is well to administer mineral materials, and the following solution of phosphate of sodium is of value: Rx Phosphate of sodium, 4 drachms; pure hydrochloric acid, 2½ drachms; distilled water, one quart. One to four teaspoonfuls of this solution may be given each day in milk, according to age. In other instances a small pinch of the following powders may be administered night and morning: Rx Sugar of milk, 2½ drachms; phosphate of lime, 2½ drachms; lactate of iron, one drachm; or saccharated carbonate of iron, lactate of iron, phosphate of lime, of each one drachm; sugar of milk, a sufficient quantity. On the other hand, Albert proposes a more simple remedy, namely, the administration of from two teaspoonsful to a tablespoonful of the following solution each day: Lime water, 6 ounces; simple syrup, 2½ ounces. In still other instances cod-liver oil is a most efficient remedy, but should it produce indigestion the following mixture of Troussseau's may be employed: Rx Pure butter, 1 lb.; chloride of sodium, 1 drachm; bromide of potassium, 7 grs.; iodide of potassium, 2 grs. This should be taken with food in the course of three or four days. Sometimes it is well to administer at the same time $\frac{1}{20}$ grain of phosphorus. Should syphilis be suspected as the cause of the rachitic tendency, it is hardly necessary to add that mercurial treatment, chiefly by inunction, should be resorted to.

Some Results of Anastomosis with the Murphy Button.—Richardson, in an interesting communication, reports three cases of lateral anastomosis by means of the Murphy button. After briefly discussing the advantages and disadvantages of this button, he states that when the patient's condition justifies a deliberate

technique he prefers to dispense with all appliances except a needle and thread. He does not regard the application of Murphy's button as brilliantly successful. His first case was one of cancerous stricture of the colon at the splenic flexure. The abdomen was opened for the relief of acute obstruction. The cæcum was found intensely distended, the sigmoid flexure collapsed. Manual exploration showed an indefinite mass in the region of the splenic flexure of the colon, probably a cancerous stricture of the colon. The patient's condition was so alarming that no radical measure seemed justifiable. The cæcum and sigmoid flexure were brought into apposition, and lateral anastomosis made by means of a Murphy button. The moment the cæcum was incised for the insertion of one half the button, there was a burst of gas and liquid faeces which flooded the whole field of operation, running among the intestinal coils and into the pelvis. The two halves of the button were applied so that the joint was not tight. This defect was remedied, after a fashion, by the application of a few sutures. Gauze drainage was employed, and good recovery followed. In two weeks there was a slight faecal discharge through the wound. The bowels began to move immediately and satisfactorily. The patient was up and able to attend to her duties in the course of a few weeks. The button was never found in the faeces. About five months later, symptoms of chronic obstruction developed. The abdomen was opened through the scar of the first incision, and under this the intestines were found firmly and densely adherent, not only to the scar itself, but to each other. The coils being separated sufficiently, exploration of the upper portion of the abdominal cavity and the region of the anastomosis was made. The button could not be felt; nor could the original disease which had been located in the splenic flexure of the colon. The cæcum was moderately distended, the sigmoid flexure collapsed. No signs of an anastomosis, and no communication was supposed longer to exist, though the coils through which the anastomosis was made were not separated, and a small communication might have escaped notice. A second anastomosis was made near the site of the first. This time the operation was satisfactory and the joint perfect. Prompt recovery followed. The bowels moved freely and regularly, aided by

cascara sagrada. The patient was discharged entirely relieved of her obstructive symptoms in a month. The button never appeared. She remained well for four months, when she was seized again with acute obstruction, causing death. Autopsy showed both buttons free in the transverse colon. Both anastomoses were still patent. Cause of death was internal strangulation. Constriction of the colon was found at the position of the splenic flexure. This had narrowed the lumen of the tube to less than the size of a lead-pencil. Both anastomoses were abundantly large. Richardson also reports the case of a man, aged sixty-three years, with cancerous stricture of the pylorus, symptoms dating back nine months. Anastomosis was made between the jejunum and the posterior wall of the stomach, the joint being perfect. The operation lasted about five minutes, and was in every way satisfactory. The patient perished with symptoms of peritonitis six days after operation. At autopsy the ring of gastric mucous membrane, compressed by the button, was necrotic; the gangrenous process had, moreover, extended into the stomach wall. The gastric contents had escaped and caused a fatal peritonitis.

Boston Medical and Surgical Journal.

DR. SCHNEIDEMAN has recently seen two instances of pretty severe intoxication from the use of daboisin as a mydriatic; both occurred in children, who, indeed, with women, are most susceptible to this effect of the drug. The strength of the solution used was $\frac{1}{4}$ grain to the fluid drachm. In view of the liability to toxic effect in such patients, it is better to reduce the strength to one-half that mentioned, even at the risk of incomplete mydriasis in some cases, requiring instillation to be continued for several days. The portion of the drug which is responsible for this unpleasant action is not that fractional part which gains access to the anterior chamber and causes the desired paralysis of the accommodation or dilatation of the pupil, but that which is absorbed by the conjunctiva and lachrymal tract, and is thus worse than wasted. A single small drop carefully applied to the upper or lower margin of the cornea is effective as a cycloplegiac; momentary eversion of the lower punctum offers an additional safeguard against absorption into the general circulation.

Philadelphia Polyclinic.

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A CLINICAL LECTURE ON HERNIA IN CHILDHOOD.

BY
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GENTLEMEN.—The subject of "Hernia in Childhood" is one of such great practical importance to those who are engaged in general practice, that although it may seem to have been worn threadbare, I would venture to draw your attention to it again to-day. Before passing to a brief consideration of the different forms of hernia which occur in young children; let me remind you that the treatment of a hernia is often not completed with the mere application of a truss. There can be no doubt that very frequently flatulence and abdominal distension tend to relieve themselves by the yielding of any naturally weak spots in the abdominal wall, such as the umbilicus and inguinal canal. Moreover, flatulence leads to screaming, and thus another potent cause of hernia is brought into play. We are, therefore, not surprised that hernia is particularly common in bottle-fed children, and in those who are put to the breast "whenever they cry," and it is obvious that attention to those matters, including the prevention of constipation, is a very important part of the treatment. I need not trouble you with more than a mere reference to the importance of coughing in the same connection, and all of us are familiar with cases in which a hernia has come down during the violent straining of pertussis. A point to which, however, I would draw your attention is the relation of hernia to phimosis. That the two conditions are frequently present together is certain, but that this is in the majority of cases more than acci-

dental is to my mind very doubtful. It is not remarkable, seeing how common are both hernia and phimosis, that they should frequently be present in the same child, and that in examining the child for the one affection the other also is noticed. Further, it is interesting to remember that hydrocele, which is very common in young children, is also frequently associated with phimosis, and that by some writers this relation also appears to be regarded as something more than accidental. It is highly probable, however, that the proportion of ruptured children who have a degree of phimosis requiring circumcision is no greater than that of children not so affected. It is difficult to understand how a tight prepuce can cause a hernia except by occasioning actual straining during micturition. In my experience this is very unusual, and I believe that obstruction to the escape of urine, as by "ballooning" of the prepuce, is quite rare. It is needless to say, however, that circumcision should be recommended if a child with hernia also has phimosis; but whether the operation can be regarded as part of the treatment of the hernia seems doubtful.

Passing now to the common varieties of hernia met with in childhood, we are concerned practically with two only — inguinal and umbilical. Femoral hernia in young children is extremely rare, probably because the crural canal is not the seat of such changes in the development of the abdominal wall as render the umbilicus and inguinal canal weak spots. In an adult female there is always a considerable probability that a hernia in the groin is of the femoral variety, but although inguinal hernia in male children is far more common than in female, yet when a hernia in the groin does occur in a female child it is almost always inguinal.

Speaking of hernia in the female, Macready says: "During the early years of life, inguinal hernia is found almost alone; but in the child-bearing period (16—50) the femoral cases are nearly equal to the inguinal, and after fifty the femoral are slightly in excess." Among the last

6000 surgical cases which have come under my care in the out-patient department of the Victoria Hospital there has not been a single case of femoral hernia.

Umbilical hernia may be congenital, but we will confine our attention to that form which occurs so very frequently in children of either sex during the first few months of life. All degrees are met with, from a slight "starting of the navel" up to the formation of a considerable globular tumour, which becomes very unpleasantly tense when the child screams. The hernial aperture in such cases is usually a round opening in the umbilicus large enough to admit the tip of the little finger. It is scarcely necessary to say that these herniae almost always become cured spontaneously during the first year or two of life, and it is well known that the affection is scarcely ever seen in young adults, although it again becomes common after middle age, especially in females. The herniae occurring under these circumstances have, indeed, no connection with the infantile variety, and as a matter of fact differ from it in their exact relations, for they usually protrude through the linea alba immediately above the umbilicus. A case such as I have lately seen, in which a man of sixty-five had had a true umbilical hernia since infancy, is very exceptional. In treating an umbilical hernia in a child, attention to the dieting is most important. To assist the natural tendency to spontaneous cure, the hernia should be retained by applying a simple strip of adhesive plaster across the front of the abdomen. The popular practice of applying a tight binder in these cases is not only useless, but actually harmful, for if it is tight enough to retain the hernia it must exercise very undue pressure on the abdomen, and may possibly cure the umbilical hernia at the expense of producing another in the groin. Buttons and pads of various kinds, intended to exert pressure on the umbilicus, may all be discarded as perfectly useless.

Strangulation of any form of hernia in a young child is very unusual, but in umbilical hernia it is particularly rare. This is a little remarkable when one considers how sharp and comparatively unyielding are the edges of the opening through which the hernia escapes. Personally, I have only once seen herniotomy performed for the relief of a strangulated umbilical hernia in a young child. The case was under the care of my

colleague, Mr. Bilton Pollard, at University College Hospital. The child was two months old, and the hernia had been strangulated for about 24 hours, the gut was swollen and congested, and the sac also contained a drachm of blood-stained fluid. From what has been said, it follows that operation for the cure of an umbilical hernia in a child is one that is very rarely indeed called for.*

Passing now to the far more important subject of inguinal hernia, it may be well in the first place to consider briefly the other conditions in a male child, which are most frequently mistaken for a rupture. Of first importance in this connection is hydrocele. Without troubling you with the well-known differences between an inguinal hernia and an ordinary hydrocele of the tunica vaginalis, it may be well to point out that real difficulty should only occur in those instances in which the narrowed upper end of the hydrocele sac passes upwards beyond the external abdominal ring into the inguinal canal—the so-called *infantile variety*. The upper limit of the swelling is then out of reach, and the resemblance to a hernia is at first sight close; and although a hernia is almost invariably reducible, it may be no easy matter to demonstrate this in a screaming infant. The value of observing the completely translucent nature of the swelling is in most cases considerable, for although a hernia in a young child may allow a little light to pass through it, its behaviour in this respect is very different from that of a thin-walled hydrocele. It may be noticed also that as the abdominal muscles contract in the act of screaming, the hydrocele is drawn upwards by the cremaster, whilst the hernia descends lower into the scrotum as it increases in size. In those cases of hydrocele in which the fluid is reducible by reason of a persistent communication between the tunica vaginalis and the peritoneal cavity—the variety to which the unfortunate name "*congenital hydrocele*" has been given,—the way in which reduction occurs, taken with the other features of the swelling, will serve to indicate its true nature. Of more practical importance in this connection is the so-called *encysted* hydrocele of the spermatic cord, in which we have to deal with a small oval cyst lying in front of the cord, usually just below the external abdominal ring. As a rule, the spermatic cord can be defined above the swelling,

and thus the distinction from hernia is easy. This, however, is not always so, and thus some difficulty in the diagnosis may arise, especially as the swelling may seem to be reducible; but even then it will be noticed that on drawing the testicle downwards, the cyst is drawn down after it. It is interesting to note that occasionally this variety of hydrocele forms so rapidly that the case is mistaken for one of strangulated hernia. Within the last few weeks a child was brought to the hospital with an encysted hydrocele presenting this peculiarity, and a medical man had been on the point of undertaking the operation for strangulated hernia, when he altered his mind and sent the child here. I may digress for a moment to mention that this acute hydrocele of the cord occurs also in the adult. Thus Curling records a case in which he was summoned to the London Hospital to operate on a case of strangulated hernia. He found the lad with "an anxious countenance and affected with nausea." The supposed hernia was, however, quite translucent, and was at once diagnosed as an acute hydrocele of the cord. "By the application of leeches and ice to the tumour, and the administration of calomel and opium, all the symptoms were relieved." A few years ago a man attended from the Military Exhibition complaining of nausea and a painful lump in the groin, which he had noticed only a few hours previously. Strangulated hernia was diagnosed, and the man sent to St. Thomas's Hospital, where a further examination showed that the supposed hernia was an encysted hydrocele.

The next point in the diagnosis to which I would refer is the relation between a retained testis and an inguinal hernia in a child. A testicle retained in the inguinal canal, or projecting just beyond the external abdominal ring, will obviously present a close superficial resemblance to a small hernia, but the mistake of confusing the two conditions will not occur if the rule is always followed of observing whether both testicles are in their normal position in the scrotum when examining a swelling in the groin of a child. But the matter does not end here, for very often a testicle retained in the inguinal canal is associated with a hernia. This is doubtless due to two causes: first, the canal under such cases is necessarily imperfectly closed; and secondly, as a general rule the tunica vaginalis of a retained testis communicates freely with the

peritoneal cavity. It is impossible here to go more fully into this subject, and it must suffice to remind you that an imperfectly descended testis is liable to sudden twisting of its spermatic cord resulting in rapid swelling of the gland, and the production of symptoms which both locally and constitutionally may closely resemble those of a strangulated hernia; and further, that in some rare instances much difficulty has arisen in the diagnosis of a case in which a small strangulated hernia has been more or less hidden by a testis retained in the inguinal canal. With the diagnosis between swellings of the testis itself and a scrotal hernia we need not now concern ourselves, except to mention that cases occasionally occur in which the symptoms of acute inflammation of the testis in a child have been seriously increased by taxis employed to it in the belief that it is a rupture.

As concerns the hernia itself, it may be remarked that it is almost invariably reducible, in this respect differing from inguinal hernia in adult males. This is doubtless in part due to the comparative infrequency with which the imperfectly developed omentum of a child enters the sac, for it is generally recognised that the commonest cause of irreducible hernia in the adult is the presence of adherent omentum. As a matter of fact the small intestine usually forms the sole contents, although on the right side the cæcum is probably not uncommonly present, and has, indeed, been met with in certain cases in which operation has been undertaken for the relief of strangulation.

The important question to which we must now pass concerns the treatment of an inguinal hernia occurring in a child a few weeks or months old. I need not repeat what has already been said with regard to certain general points which should never be neglected, but pass at once to the local treatment of the hernia itself.

In very young children considerable difficulty is often met with in the use of a truss, especially amongst the class of patients who attend the out-patient department of our hospitals, and an efficient retentive apparatus can as a rule be made with a skein of soft Berlin wool. One end of the skein is placed over the position of the hernia, the skein is passed round the child's pelvis, threaded through the end which lies over the hernia, and carried thence between the scrotum and the thigh, and so upwards to the turn around the pelvis, to which

it is fixed with a safety-pin or by knotting it. In this way a twist of soft wool is made to exert firm pressure on the inguinal canal. The mother is instructed to prepare two or three similar skeins in order that they may be changed for washing. In doing so, she is told to replace the wool "truss" by a clean one as quickly as possible, and to keep the hernia back by pressure with her finger during the process. By the use of this simple means, which is, however, not invariably successful, the chafing of the skin, the swelling of the scrotum, and even the orchitis, which are apt to be produced by any other form of truss, are avoided, and it occasionally happens that the use of the latter is rendered altogether unnecessary, for if the treatment is commenced shortly after the hernia is first noticed, and the latter can be kept back for a couple of months, it is not uncommonly once and for all cured.

If, however, this is not the case, and the child has reached the age of perhaps six months, some ordinary form of truss should be advised. Much difference of opinion exists as to which is the most convenient pattern, but it may certainly be said that no form in which the pressure is exerted with a spring surrounding the pelvis is suitable, for it is almost certain to cause troublesome irritation of the skin. Perhaps the best is a soft rubber horse-shoe-shaped truss, the limbs of which lie one over each inguinal canal, and are prolonged into soft rubber bands which take the place of the perineal straps of an ordinary inguinal truss. The truss is attached by a flat india-rubber band encircling the pelvis. This pattern has the additional advantage that it is equally applicable to single and double herniae; it should be worn constantly, and, if removed, applied again as quickly as possible. If it is carefully dried when the child is washed it is unnecessary to remove it for that purpose, but the mother should be warned to dry the truss and the skin beneath it very thoroughly, and to use some simple dusting powder, such as boric acid, to prevent excoriation. The constant application of the truss is the one great point in the treatment, for it must clearly be remembered that the purpose for which it is used is quite different to that for which a truss is employed in adults. In the latter it is simply a palliative measure, whilst in the former it is hoped to be curative, for there can be little doubt that the commonest cause of inguinal

hernia in a young child is the persistence of part of the funicular process of the peritoneum, which should have become closed before birth, and that if this peritoneal sac be kept empty for a few weeks or months it undergoes the same changes after birth which it should have undergone earlier, and the hernia is radically cured. The mother should be reminded that every time the hernia comes down she is practically starting the treatment afresh, and that with each recurrence the chance of obtaining a cure by this simple means becomes proportionately less.

In children of over two years old, an inguinal truss of the ordinary pattern can be employed, but it is advisable that it should be covered with india-rubber instead of with leather, otherwise the leather soon becomes soiled and the spring rusty and spoiled. In the cases most commonly met with in which the treatment of the hernia is commenced during the first two or three months of life the cure by the use of a truss may be anticipated before the child is a year old, at about which time as a general rule the truss should be left off, but with the slightest sign of weakness in the part it should be re-applied for some months. Practically speaking, all herniae occurring in children under twelve months may be reasonably hoped to be capable of cure by the use of a truss, but after this age one should speak much less hopefully in giving a prognosis.

We now have to consider shortly the matter of recommending operation for the cure of an inguinal hernia in a young child, and in the first place let me remind you, and it, indeed, follows from what has been said, that it is only in a very small minority of cases that any operation is called for. This fact seems to require emphasis because one cannot doubt that there is rather a tendency at the present day to recommend operative interference in cases of hernia in children, perhaps rather more freely than the circumstances of the case require. Under no circumstances should operation be undertaken in a child of under two years in whom the hernia is efficiently retained by the use of a suitable truss. In some instances, however, the hernia is of unusual size, the inguinal canal and the external abdominal ring are proportionately large, and by no means can the hernia be kept properly reduced. Under these circumstances, the chance of a cure

being effected by the use of a truss is practically *nil*, and, moreover, in many instances the hernia appears to be a source of considerable inconvenience to the child, and as a result constant fretfulness and screaming are induced, and the hernia correspondingly gets worse. In such cases, in children of even a few months old, radical cure by operation may at times justly be undertaken. In children of two or three years old and upwards, in whom, although the hernia is kept back efficiently by the truss, but comes down whenever the truss is removed, even though the instrument has been worn regularly for a year, the chances of a permanent cure are, as we have seen, small. Under these circumstances, the probabilities are so considerable that the truss will continue to be necessary, that there can be little doubt that, if the general health of the child is good, the proper treatment is to recommend a radical operation.

The rule which guides us is a little different to that which is followed in the treatment of adults. In the latter case the rule must still hold good, that under ordinary circumstances a patient who has a hernia which is efficiently kept back by a truss should not be urged to submit himself to an operation, although the surgeon is justified in performing it at the patient's own wish, when the facts of the case have been clearly put before him. In a child, however, the existence of a hernia necessitating the use of a truss may later on prove such a serious drawback, and the results of operation are so favorable, that it should certainly be recommended.

I have so far been speaking only of reducible hernia, and, as we have seen, nearly all cases come under this heading. It is needless to say that in those rare instances in which a child is affected with an irreducible hernia, operation must under ordinary circumstances be strongly recommended. Nor have I spoken of strangulation, which, as we have seen is very unusual. Not uncommonly in a screaming baby it is impossible to reduce a tense inguinal hernia, and a slight degree of strangulation may doubtless occur, but almost invariably with the relaxation of the abdominal walls induced by the administration of a few drops of chloroform, the hernia slips back almost without the application of taxis. If this do not occur, operation must be proceeded with without delay, and will be com-

pleted by proceeding to the necessary steps for the radical cure now to be described.

The following method will be found efficient for the radical cure by operation of an inguinal hernia in a child: A short incision is made over the inguinal canal extending down to the external abdominal ring, but *not into the scrotum*. The ring is exposed, and the external oblique aponeurosis is slit up in the line of the skin incision sufficiently to open the whole length of the inguinal canal. The upper part of the sac in the canal is now clearly defined by dividing the tissues which still lie over it, particularly the well-developed cremaster. The sac itself is then carefully separated from the constituents of the spermatic cord, which are often rather firmly adherent to it posteriorly, and it is then isolated to the extreme upper end of the inguinal canal, and after being opened in order to make sure that it is empty, it is ligatured with silk as high as possible and divided below the ligature, the ends of which are cut off short. A second fine ligature should now be placed on the sac below the point of division, but the body of the sac which lies in the scrotum is left undisturbed. This considerably diminishes the disturbance of parts occasioned by the operation, and the empty sac which is left behind in the scrotum is never a source of any subsequent trouble. The divided aponeurosis of the external oblique is now readjusted with fine silk sutures, the last two or three of which are placed deeply through the pillars of the external abdominal ring so that the latter is reduced to such a size as just to admit the tip of the little finger. All bleeding having been thoroughly arrested, the edges of the skin are brought together with horsehair sutures, and a simple antiseptic dressing is applied. The latter should be small, and applied with a spica bandage; a large dressing in a young child is best avoided, as it is more likely to become soiled with the urine. Between the last turns of the bandage it is convenient to place a piece of gutta-percha tissue in order to protect the deeper part of the dressings. As a general rule, primary union is found to have occurred, and the sutures can be removed at the first dressing on the ninth or tenth day, but the child should be kept on its back as much as possible for four weeks with a small pad

firmly applied with a spica over the seat of the operation. A pad should be kept constantly applied for five or six weeks, after which it may be dispensed with altogether, and the use of a truss after the operation is not only useless but probably harmful. The only two points in the operation which need be insisted upon are the importance of keeping the incision out of the scrotum, and the advantage of slitting up the external oblique in order that the sac may be ligatured as high up as possible, for this must be regarded as the essential point of the operation. It certainly is so if we consider the most important cause of the hernia to be the existence of an already formed sac which merely requires removal to effect a cure. Perhaps the operation for inguinal hernia which at the present day has gained most favour is that which was introduced a few years ago by Bassini of Padua. The essential feature of this operation is that it endeavours to restore the natural valvular arrangement of the inguinal canal by closing the latter in two separate layers, one behind the spermatic cord and the other in front of it, instead of in one layer in front of the cord, as is done in most other methods. After the external oblique has been slit up and the sac of the hernia dealt with, the spermatic cord is raised from its bed and the arched borders of the internal oblique and transversalis, passing to the conjoined tendon, are drawn downwards and attached with a series of sutures to the deep aspect of Poupart's ligament. The cord is then dropped back into position, and the edges of the divided aponeurosis of the external oblique sutured over it, the suturing being continued along the pillars of the abdominal ring. This operation is probably the most efficient which has been introduced for the radical cure of an inguinal hernia in the adult; but in a child, unless the inguinal canal is unusually dilated, the disturbance of parts which the operation necessarily involves is undesirable, and the simple method above described gives results which leave little to be desired.

Considerable difficulty may occur in treating a case in which an inguinal hernia is associated with an imperfectly descended testis. If the testis is not sufficiently high up to prevent the application of a truss to the hernia above it, this should of course be done, and thus the hernia may be cured and the testis may so far descend that its position

becomes a matter of no inconvenience. Frequently, however, in a child a few months old the hernia cannot be reduced without taking the testicle with it. As a rule it is then wise to do nothing for a while, unless the hernia is increasing in size, in the hope that the testicle may come down a little, and so allow a truss to be applied to the hernia. If this does not occur it is probably better to resort to operation than to adopt the alternative of keeping both the hernia and the testis retained by the use of a truss. In older children one should not hesitate to advise operation. It is sometimes necessary to remove the imperfectly developed gland, but as a rule an attempt should be made to fix it as low down in the scrotum as possible. The application of a specially constructed truss in these cases with the object of retaining the hernia, and pushing down the testicle, is usually a practical impossibility.

Little need be said concerning the treatment of inguinal hernia in the female. As a general rule the treatment which has been described above will readily produce a cure, and operation is rarely called for. It is interesting to remember that not very rarely the ovary on one or both sides may pass along the inguinal canal and lie just outside the external abdominal ring. As a rule in such cases reduction can be effected, and a cure obtained by applying a truss. Recognition of the condition is easy, for the ovary can be felt as a small oval body slipping about freely beneath the skin.

Occasionally in older children operation is required for this condition; and in one case of double ovarian hernia in a girl of six years under my care, it was found necessary to remove one ovary which could not be satisfactorily reduced, whilst on the other side the ovary was reduced, and the sac ligatured and removed.

In rare instances strangulation of an ovarian hernia has been met with, but it is highly probable that some of the cases regarded as of this nature were rather examples of torsion of the ovarian pedicle, a condition which has recently been met with by Mr. Lockwood and Mr. Owen. This remarkable accident, followed as it is by swelling and haemorrhage into the ovary, is evidently closely allied to the sudden torsion of the spermatic cord of an imperfectly descended testis in the opposite sex.

A STUDY OF APPENDICITIS.

By PROFESSOR DIEULAFOY.

(Bulletin de l'Académie de Médecine.)

(Concluded from p. 317.)

Onset of Appendicitis.—How does appendicitis show itself? Between an insidious, apyretic onset and an acute, pyretic, and very painful onset, one may observe all the intermediate stages.

Usually matters take place as follows: after a day or two of loss of appetite, constipation, rarely of diarrhoea, the patient feels a sensation of weight, of tension, of pain in the right iliac fossa; sometimes some nausea or vomiting accompanies this condition. If there is fever it is slight and insignificant. Without waiting for his doctor the patient commences to administer to himself a purgative or enema; he is conscious that the intestinal functions are not normal. The doctor comes; the patient speaks chiefly of his constipation, of the furred condition of his tongue, of the loss of appetite, of the nausea, and he calls attention to the painful right iliac fossa. "Certainly," says the doctor, "I find a little fulness there—a little tension," and if the doctor in question still believes in typhlitis he makes a diagnosis of stercoral typhlitis, of cæcal engorgement, and he prescribes "a good purgation," a blister or leeches.

Under other circumstances, the pain of the appendicitis occurs almost without prodromata: it is the dominant element, it quickly reaches a sharp intensity; it localises itself at first in the right iliac fossa, but it spreads in various directions. Vomiting, especially bilious vomiting, is frequent. The doctor comes, and if he is imbued with the ideas of Palamon, he declares, after having examined his patient, that it is a case of "Colic of the appendix;" that is to say, that a calculus of the cæcum is passing into the canal of the appendix, and that it sets up in its passage very sharp pains, usually accompanied with nausea and vomiting. The doctor orders antipyrine, chloral, administers injections of morphine, which procure for the patient a deceptive calm. If the pains cease or diminish in intensity it is said that the colic of the appendix has terminated, and that the calculus has fallen back into the cæcum; if the pain reappears, one

supposes that the calculus, or other calculi, have become engaged in the appendix, and one maintains the diagnosis of colic of the cæcum and appendix.

Well, I will speak out plainly. It is a false and dangerous theory. Rochaz has demonstrated that calculi do not wander thus into the narrow canal of the appendix; the pains of appendicitis are not therefore the pains of a calculous migration, "colic of the appendix" does not exist. This term perpetuates an error—it ought to disappear; it is altogether as false to speak of colic of the appendix, as it is misleading to speak of typhlitis; it is with these terms, it is with these false theories, that one loses precious time. They say we have nothing to fear; we are only in the period of colic of the appendix; we shall see later on; we shall come to some decision if peritonitis sets in. But it is with such reasoning that one lets the patient die of peritonitis or of subacute peritoneal septæmia! One puts off to the next day, or the next after that, an intervention which does not appear to be indicated so long as one still believes oneself to be dealing with "Colic of the appendix," and one realises later, sometimes too late, that what one has taken for so-called colic of the appendix was the pain due to inflammation of the appendix; and the proof of this is that these pains exist in cases of non-calculous appendicitis, and they exist also when the canal of the appendix, narrowed or obstructed, would allow no calculus, small or great, to enter or to leave it. The false theory of colic of the appendix is then condemned, and the term itself, I repeat, confirms an error that ought to be erased from our nomenclature.

To what then are due these pains, sometimes so sharp, of the first phases of appendicitis? There cannot be for a moment any question of perforation of the appendix, or of peritonitis.

These pains, like the symptoms of the onset,—abdominal tension, nausea, constipation, vomiting gastric and biliary,—are the consequences of the transformation of the canal of the appendix into a closed cavity. The same thing occurs in the appendix as occurs in the cavity of the tympanum transformed into a closed cavity by obstruction of the Eustachian tube. When the tube is obstructed the microbes of the middle ear have their virulence increased, otitis is set up, and with this start the pains, sometimes so violent, which shoot in various

directions. Otitis often terminates without perforation of the tympanum and without other accidents, it is sufficient to ensure this that the Eustachian tube becomes permeable again, whether artificially or naturally ; then the obstacle being removed, the free evacuation of the infective products remove the dangers of a closed cavity. But if the obstruction persist, the microbes imprisoned in the cavity may have their virulence increased to such a point that all complications are possible ; there is perforation of the tympanum analogous to perforation of the appendix ; there is phlebitis of the lateral sinus and of the jugular vein analogous to phlebitis of the appendix ; there is meningitis, so common in children, analogous to peritonitis round the appendix ; there are the abscesses in the neck, and even abscesses at a distance in the cerebral hemisphere of the opposite side, analogous to distant peritoneal abscesses, or abscess of the liver.

The complications are thus absolutely analogous in otitis and in appendicitis. It is the closed cavity and the inflammation of the appendix which determine the sharp abdominal pains wrongly mistaken for the colic of a migrating calculus ; it is the closed cavity and the inflammation of the appendix which give rise to the digestive troubles,—the nausea, the vomiting as in an internal strangulation ; it is the closed cavity and the infective appendicitis which determine sometimes in a rapid manner, the toxic symptoms, the depression, the collapse which constitute a large part of the gravity of the illness ; it is the closed cavity and the infective appendicitis which are often productive, before any peritoneal lesion, of infective inflammation of the liver, of areolar abscess of this organ, and of general infection.

Thus, from the onset, *before the advent of any peritoneal lesion*, inflammation of the appendix is there, menacing, and revealing its presence by the troubles which I have just recounted ; they must be blind who do not see this, and who, still trusting in an antiquated formula of typhilitis and of colic of the appendix, satisfy themselves with phrases, and temporise when they ought to act. Now to avoid all mistake, I propose for this stage the denomination of "*accès appendiculaire*."

It is important, then, to depict with certainty this appendix-seizure ; it is important to make, from onset, the *diagnosis* of appendicitis. Most frequently the pains in the appendix are at the maxi-

mum in a region situated in the centre of a line drawn from the umbilicus to the anterior superior iliac spine. This is the point of McBurney. It is necessary to examine methodically this painful region, and then, although the pains radiate to other points, one can, by palpation, by methodical pressure, provoke, excite, or increase the pain in the appendicular zone. At this level, the adjacent muscles contract and draw up more than at other points ; this muscular retraction is a good index. At the same place one may observe another sign on which I have especially insisted in my lectures at the Faculté ; this is *hyperesthesia* of the region of the skin corresponding to the appendicular zone. If one lightly strokes the skin of this region with the pulp of the finger or with a pencil, one observes a hyperesthesia which does not exist elsewhere. These different signs, of which the place of greatest intensity is in the iliac fossa, allow of the differentiation of the painful symptoms of appendicitis from the painful symptoms of hepatic colic. The difficulty is greater, it is true, when the appendicular lesion attacks an appendix of the ascending type, which may ascend along the posterior face of the caecum as high as the colon : even then the difficulty is not insurmountable.

Having described how appendicitis commences, let us now see what is its evolution.

The evolution of appendicitis.—Often enough, after two or three days of symptoms more or less painful, of digestive troubles more or less acute, with a fever generally quite moderate, calm is fortunately re-established, the constipation gives way, the iliac fossa is less tender, less tense, the muscular wall is less irritable, the hyperesthesia disappears, and the patient is cured of this appendix seizure.

How does this come about ? May be, with the aid of the phagocytes, the virulent enemy has been vanquished ; may be the obstruction in the canal of the appendix has yielded, the cavity is no longer closed, the free circulation of the appendix is re-established, and the appendicitis clears up as otitis clears up when the Eustachian tube regains in time its permeability. In other cases the attack of appendicitis is more serious, the pain in the iliac fossa is more intense or more persistent ; the tension, the distension of the abdomen are increased, vomiting more frequent, and yet little by little everything gets again into its proper

order ; but then convalescence is slow, for a long time the patient retains in the iliac fossa a reminiscence of his appendicitis ; he is constipated, he feels some apprehension about hunting, about mounting a horse, about fencing, and if he decides to be operated on later, we are able to make out some adhesions of the peritoneum round the appendix, a small encysted abscess, and sometimes a threatened perforation of the appendix.

All surgeons have seen this ; I have myself observed it several times. I have had in my hands the appendix of a young man, operated on at my request by Routier in the "Necker" Hospital ; this man walked about and followed his occupation, all the while having a peri-appendicular abscess, the remains of an acute appendicitis.

In other cases the initial symptoms of the appendicitis are not excessively severe, the pains are not very sharp, the fever is moderate, one calls it truly an inflammation of the appendix of average intensity ; the digestive troubles, anorexia, constipation, nausea, alone attract the attention of the patient, who believes himself to have an attack of gastric trouble, of gastro-intestinal indigestion. Three or four days pass, the abdomen becomes distended, the pain, at first localised in the right iliac fossa, becomes generalised without increasing in intensity, the aspect is altered, the pulse becomes quickened, peritonitis is evident, fluid accumulates in the peritoneum, but one does not know at what moment the peritoneal stage followed upon the stage of appendicitis ; the temperature falls, a slight hiccough comes on, the patient falls into collapse, becomes cold, and dies. I allude to a case to which I was called in consultation last October with Pinard and Segond.

There are cases, on the other hand, where the symptoms develop much more rapidly. Whether or no there be perforation of the appendix, the peritoneal lesions come on early, are very septic, and, if the operator does not intervene quick enough, the patient succumbs on the third or fourth day. One finds, at the operation or at the autopsy, not a peritonitis with abundant purulent fluid, but a sort of subacute peritoneal septicaemia, with some adhesions and a small quantity of pinkish fluid of excessive virulence. I have recently observed an example of this kind. On the 28th of November of last year, in a family in which I have long been the medical adviser, I was called

to a lady of 72 years, who had had during the night sharp pains in the stomach. Two days previously this lady had been to her dentist, and on the evening of the day before I saw her she had felt very well. I examined her in the morning, and I made out that the pains had very distinctly their maximum of intensity in the appendicular zone ; the temperature was normal, but the pulse was small, and the aspect did not satisfy me. On arriving at the "Necker" Hospital, where I knew I should find my colleague, Routier, I asked him to be so good as to undertake the operation. During the day, on our visit to the patient, the temperature was rising, and had reached 38.5° (101.3° F.), the pulse was not good, the iliac fossa was painful, thickened, and yet the beginning of the attack was within the last twenty-four hours.

The operation was done the same evening—fortunately, for the patient was already in full peritoneal septicaemia ; the appendix was turgid and congested. I pass over the details and the events following the operation. This patient, 72 years of age, was completely cured. What would have happened if, trusting on old theories, I had waited till the morrow to decide on operation ? The patient would certainly have succumbed, as did a patient, herself also 72 years of age, whom I saw a few days later with Planchon and Monod.

I have not yet exhausted all the varieties of acute appendicitis ; in some cases, probably sufficiently rare, the gravity of the mischief does not consist only in the peritoneal complications, it consists chiefly in the infective inflammation of the appendix itself. I was able to examine the appendix of a young girl, sent to me by Routier ; this appendix, very long, very voluminous, very hard, as if in a state of erection, contained two calculi, and it was over these calculi, free in the closed cavity, that the canal of the appendix had been obstructed by the swelling of the walls of the appendix. The *Bacillus coli* and Streptococci were the virulent agents in this closed cavity. There was neither ulceration nor gangrene, nor perforation of the appendix. The following is the history which Routier gave me. "After two days of malaise, a young child of eleven years fell ill on a Saturday morning. She had nausea, green diarrhoea, and pains in the right side of the abdomen. One thought of appendicitis, and ordered ice to the stomach and opium ; during the night vomiting

came on. The following morning, Sunday, the tongue was dry, red, parched, the expression anxious, the stomach much distended ; the pulse was 120, the temperature, slightly raised, was at 38.5° (101.3° F.) This same Sunday morning, Routier saw the little patient at 11 o'clock. He found the condition very alarming ; all the symptoms were accentuated, and he formed the diagnosis of generalised peritonitis, consecutive to a perforative appendicitis. He performed an operation at noon, and to his great surprise he found only some very slight traces of peritonitis ; the peritonitis was only in the initial stage ; but the appendix was enormous, it was three times its normal size, it was as hard as wood, it was neither gangrenous nor perforated."

After the operation an arrest was produced, the vomiting ceased, the pulse improved. But this amelioration was of short duration, for the inflammation and infection were already generalised, they had done their work, and twenty-four hours later, in spite of the cessation of the symptoms of appendicitis, the child succumbed in a state of collapse, poisoned by the virulent products of the closed cavity.

This proves that in appendicitis it is not only perforation which is to be feared, it is not only peritonitis—there is also the inflammation of the appendix itself. Appendicitis is not only an inflammatory lesion, it is *infective* ; it may have by itself a considerable part in the accidents that arise, and this from the onset of the malady, from the time when the closed cavity is constituted.

How far we are from the artificial division imagined by Talamon ! According to this author, whose words I cite textually, "there are habitually in appendicitis two very distinct periods, the preparatory period which precedes perforation, and which I propose to call the period of colic of the appendix, and the phase of peritonitis which follows the rupture of the appendix." *

Each one of these assertions contains an error, and it is time to reassert the facts. Imbued with such theories one might believe, and wrongly, that peritonitis only comes on when the appendix is perforated, but a good number of instances have been cited—and I have confirmed them—where peritonitis in all its varieties, generalised or localised,

Talamon. ' Appendicite et perityphlite,' p. 102. Coll. Charcot-Debove.

may exist, while the affected appendix is not perforated at all.*

Clinical experience is thus in accord with the experiments of Klecki. Further, one might think wrongly that the stages of peritonitis and those of appendicitis are distinctly marked ; but it is often impossible to know at what moment the peritoneal complications follow on the symptoms due to the appendix. These symptoms and complications are, so to say, fused and mixed together. The pains, the vomiting, alimentary and bilious, the condition of the temperature and of the pulse, the constipation and the tympanites of the abdomen, all these signs exist with or without the peritoneum being involved ; and it is certainly difficult in many cases to know at what moment peritonitis declares itself. There are, then, not always, we must remember, two distinct periods in the evolution of appendicitis and of peritonitis.

To recapitulate, when an appendicitis commences, whether its features appear benign or severe, one never knows what surprises are in reserve. The appendix-seizure stage, the "*accès appendiculaire*," may in itself comprise the whole malady ; it may get well with or without adhesions.

Under other circumstances appendicitis leads to peritonitis ; in such cases the appendix is often perforated, necrosed ; but sometimes also peritonitis comes on when the appendix is neither perforated nor necrosed.

The peritonitis may take on the most varied forms ; it is generalised or partial, it is widespread or very limited. The direction normally occupied by the appendix before the illness (descending type, ascending and retro-cæcal type, inner lateral type), and the adhesions, old or recent, are some of the conditions which explain the localisation of the peritonitis, and of its tendency to become encysted. Thus we get partial ileo-inguinal peritonitis, retro-cæcal, peri-rectal, peri-umbilical peritonitis, &c., forming small pits, purulent foci, and peritoneal abscesses. These encysted peritoneal abscesses may open spontaneously into the cæcum, as I saw in a case with Pozzi and Weil ; into the colon, as I have observed recently with

* Monod, "Contribution à l'étude des appendicites," 'Bulletin de la Société de Chirurgie,' 1895, p. 497; Reynier, "Appendicite avec appendice non perforé," Seance de la Société de Chirurgie du 4 Mars, 1896 ('Presse Médicale,' 1896, No. 20, p. xcvi).

Barbe and Routier; into the rectum, into the vagina,* into the bladder, into the bronchus in the manner of a vomica.† These spontaneous openings are most frequently followed by recovery.

The gravity of the peritonitis of appendicitis is determined by the *double infection* which is elaborated in two centres, the appendix and the peritoneum. I have said that of the number of microbes which we have found in the appendix, the *Bacillus coli* and the streptococcus appear to be dominant. The microbes of the peritoneum in cases of peritonitis from appendicitis have been well studied by Macaigne, who has had the extreme courtesy to give me the unpublished results of his researches on this subject. Macaigne has studied bacteriologically eighteen cases of suppurative peritonitis following on appendicitis and operated on by Monod. The pus of this peritonitis contained generally a large number of microbes, micrococci, streptococci, *Bacillus coli*, staphylococci. In the majority of cases one observed most prominently two microbes, the streptococcus and the *Bacillus coli* (the same which we had found with Kahn in the pathological fluid of appendicitis). These microbes were in numerous colonies, and one or other predominated, according to the case.

These microbes then reach the peritoneum with a virulence already much exalted, and it is certain that their *degree of infectiveness* plays a considerable part in the severity and the features of the peritonitis which follows. Sometimes they are sent into the peritoneum through a perforation or tear in the appendix, sometimes they reach the peritoneum through the walls of the appendix altered by disease, but not perforated. Here also clinical experience is in complete accord with the experiments of Klecki.

I wish now to say a few words on the extra-peritoneal complications which may result from appendicitis. I have chiefly in view hepatic inflammation, conveyed by the veins of the appendix with or without phlebitis of the appendix, hepatic infective inflammation, which shows itself pretty frequently in the formation of areolar hepatic abscess, accompanied by complications always fatal. These

facts have been well studied by Achard.* They prove that one may succumb to a hepatic infection, and to a generalised infection with its origin in the appendix, even when peritonitis is not "on the cards," even when the patient seems as though he ought to recover from his appendicitis.

Such are the many and terrible mischiefs of appendicitis; hence one understands that the question of treatment demands serious consideration.

Treatment.—I assert with full conviction that *no medical treatment for appendicitis exists*. Medical treatment is good for nothing but to waste precious time. It is of course understood that we must relieve the patients; it is also indicated to make use of antipyrine, of injections of morphine, of applications of ice and other soothing measures, but, once more, do not let us deceive ourselves as to the efficacy of these measures; too often they make us fancy an improvement in the illness by masking the symptoms. Seeing a factitious improvement one rests content with the sweet quietude, one thinks, alas! of the so-called typhlitis, of the so-called colic of the appendix, one announces in good faith the recovery of the patient, and the patient dies because he is not operated on in time, or because he is not operated on at all.

Surgical treatment is therefore, in my opinion, the only rational treatment for appendicitis; it is the only safe treatment against its immediate accidents and the remote complications; it is the only one which obviates relapses and their consequences. One may quote to me examples of patients who have recovered without operation; certainly yes, we have seen such, but I will quote on the other side numerous examples of patients who have succumbed *classiquement*, or in the regular way, because operation has been wrongly rejected or adjourned.

It is, then, for us physicians to know how to take decisive action, it is for us to bring home to the families who confide themselves to our care the convictions which animate us. Too often we appeal to the surgeon after having long hesitated and after having tried the useless baggage of our medicinal means; it is not sufficient merely to operate, it is necessary to operate at the right time.

There is a formula I would engrave on the minds of those who could still remain hesitators: One

* Gérard-Marchant, "Appendicite par corps étrangers," *Bulletins de la Société de Chirurgie*, 1895, p. 495.

† Monod, Routier, "Discussion sur l'Appendicite," *Bulletins de la Société de Chirurgie*, 1895, pp. 500, 537.

* Achard, loc. cit.; Berthelin, "Complications hépatiques de l'appendicite," *Thèse*, Paris, 1895.

never repents of having operated in appendicitis; one often repents of not having operated or of having operated too late.

I close these considerations on appendicitis with the following conclusions:

(1) *Appendicitis is always the result of the transformation of the canal of the appendix into a closed cavity.*

(2) This transformation of the canal of the appendix into a closed cavity may take place at any point in the canal, of which the length and narrowness lend themselves so well to such a transformation.

(3) The transformation of the canal of the appendix into a closed cavity takes place by various mechanisms, which I have been able to observe and to study on appendices removed by the surgeons. Often the partial obliteration of the canal of the appendix and its transformation into a closed cavity are due to the slow and progressive formation of a calculus of the appendix, which is more or less hard, according as inorganic matters (salts of lime and of magnesia) are mixed in more or less considerable quantity with the organic and stercoral materials of the calculus. We have to do here, not with a calculus passing from the cæcum, which some have wrongly supposed, but we have to do with a true *lithiasis of the appendix* which I compare with renal or hepatic lithiasis.

(4) I have been able to demonstrate by a large number of examples, the pathogenic similarity of these three kinds of lithiasis, their coexistence in the same family, and the *heredity* of calculous appendicitis, which I propose to classify henceforth with the inheritance of gout and of arthritis.

(5) Under other circumstances, the transformation of the canal of the appendix into a closed cavity is in consequence of a local inflammation (infection) in all respects comparable with the obstruction of the Eustachian tube in the case of otitis, and to the obstruction of the bile-ducts in the case of so-called catarrhal jaundice. In some cases, also, the transformation of the canal of the appendix into a closed cavity is the slow and progressive result of a fibrous stricture comparable with the strictures of the canal of the urethra. We may add that several of these causes may be found co-existing in the same subject. Lithiasis of the appendix, and obliteration by thickening of the walls are in fact often found together.

(6) The symptoms of appendicitis, benign or grave, slight or severe, do not declare themselves until the formation of a closed cavity is accomplished. This is the "*accès appendiculaire*." At this moment the microbes of the appendix, inoffensive up to now, multiply in number and increase in virulence. The same thing occurs here as occurs in the remarkable experiments of Klecki, after ligature of a loop of intestine. It is from these experiments that I have formulated the theory of appendicitis from a closed cavity, a theory confirmed by the experiments of Roger and Josué.

(7) In appendicitis so originated, a centre of infection is established, sometimes terrible, of which the principal microbial agents are the *Bacillus coli* and the streptococcus, as I have several times confirmed with my *interne Kahn*.

(8) The virulence of appendicitis is sometimes so considerable, that the patient may succumb to the mere occurrence of an *infective appendicitis*, the symptoms of peritonitis being scarcely developed.

(9) In many cases the infective inflammation spreads from the appendix towards the peritoneum, the walls of the appendix *not* being perforated, and in spite of this absence of perforation, one may observe all the varieties of peritonitis, acute septicaemia of the peritoneum, general peritonitis, encysted peritonitis, peritonitic abcess at a distance (metastatic), and areolar abscess of the liver.

(10) Also in the cases which were considered the most classic, because they were the best known, the inflammation of the appendix leads to gangrene and perforation of the appendix, and one sees the evolution of the different varieties of peritonitis by perforation.

(11) Medicinal treatment is useless or insufficient; the only rational treatment is by surgical intervention practised *at the opportune time*.

Dermatol in Blennorrhagia.—Constantini cured four cases of acute blennorrhagia with gonococci in the pus by means of injections of water containing 2 to 4 per cent. of *dermatol* from one to three times daily. He used no other form of treatment, and recommends the method as being simple and easily applicable in general practice. Dermatol acts as an astringent and cicatrizing, diminishing inflammation, arresting the mucous-membrane secretion, and killing other pyogenic microbes that cause secondary infection. The drug has also the advantage of being neither odorous nor caustic.—*Riforma Med.*

A CASE DEMONSTRATED AT THE CLINICAL MUSEUM

BY

JONATHAN HUTCHINSON, LL.D., F.R.S.

Reported by J. T. CONNER, M.D.

A Short-limbed Dwarf: Multiple Exostoses.

THE subject was a man of deficient intellect, æt. 38, sent by Mr. Hopkins from the Cleveland Street Asylum. The limbs were considerably shortened, but the trunk and head of ordinary size, so that when seated he appeared of average height, though really only four and a half feet. There was nothing peculiar in the head or face except a heavy, dull expression. The chest was well formed, the clavicles and shoulders normal. There were two considerable exostoses at the base of the right scapula, none on the left. There was no evidence of deformity in the pelvis. From the tip of the acromion process to the external condyle measured ten and a quarter inches on either side. The humerus tapered suddenly above the condyles, which appeared rather large. The hands, which were of normal size, were bent to the ulnar side from defective length of the ulna in comparison with the radius, especially on the right side. The radius was seven and a half inches on either side, whilst the right ulna was only five and a half, and the left six inches. Both ulnæ, at their lower ends, showed abrupt enlargements. The line of the right radius, about two inches above the styloid process, presented an angular projection as if of a united fracture. This was absent on the left. The carpal end of the radius was not in the least thickened on either side.

The left lower extremity measured twenty-one inches from the external malleolus to the great trochanter, the right twenty-one and a half inches. An exostosis sprang from the inner side of the internal condyle of the right femur, apparently at the epiphyseal line. There was but little indication of any similar growth on the left. The right tibia was distinctly bent forwards, and there was an exostosis on the inner part of the head. The head of the right fibula was much enlarged, and presented an abrupt collar of elevation from the

shaft much like that on the ulna. Above the ankle-joint was the same condition of an elevated ring of bone apparently marking the line of the epiphysis. The external malleolus was not enlarged in either limb. In the left leg the head of the fibula was yet more enlarged than in the other, the enlargement passing lower down the shaft. There was an exostosis on the inner part of the head of the tibia, as in the other. The feet were normal in size and form.

The arrest of growth was no doubt due to the formation of the exostoses at the epiphyseal lines. It was remarkable that the limbs should be so severely affected, and yet the hands and feet quite normal. A portrait of a skeleton in the Dupuytren Museum, showing this in a still more extreme form, was produced. A full-sized hand was suspended by a short interval of bone from the shoulder. Such cases showed that the hands and feet developed independently of the limbs. The disease was sometimes described as foetal rickets; but Mr. Hutchinson thought it had no connection with that disease.

THERAPEUTICAL NOTES, &c.

Radical Treatment of Hypertrophied Prostate.

BY BRUNS, OF TUBINGEN.

THE treatment recommended for hypertrophied prostate is that of double castration, in preference to all others, such as ligation of the iliac arteries, unilateral castration, and ligation of the vasa deferentia.

In fifty-six cases out of one hundred and forty-eight a marked diminution in the size of the prostate followed the operation of double castration. Atrophy usually begins immediately after operation: in a few cases the results were not as good as in the others; this was due probably to the peculiar anatomical form of the hypertrophied gland. After double castration, atrophy can be said to be a functional tendency in an hypertrophied prostate; this is a reflex nervous action, since it has been proved that no anatomical relation exists between the prostate and the testicles.

In view of the relation between the hypertrophied

prostate and the condition of the bladder, hypertrophies can be divided into three groups :

(1) Hypertrophy with retention ; results are good in these cases, micturition becomes less frequent, tenesmus disappears ; at times the patient can urinate voluntarily.

(2) Hypertrophy with retention, wherein catheterization must be employed.

(3) Hypertrophy with retention, co-existing with diuresis ; in some of these cases the patient can pass his water voluntarily, because of the excessive secretion of urine ; in other instances, catheterization is necessary, but is facile, and seldom required.

Only one feature of the results following double castration is objectionable, namely, the promotion of premature senility, when practised upon men not over sixty years of age.

The author thinks that resection of the *vasa deferentia* is, or rather will be, only practicable and preferable to all others when it shall have been proved that the functional after-results are analogous to those following double castration.

Annales des Maladies des Organes Genito-urinaires.

Bartholinitis.—Bergh, in discussing inflammation of Bartholini's gland, shows that the inflammation is principally located in the duct of this gland. In addition to specific inflammation, there are other forms, namely, those due to streptococci and staphylococci. Usually the inflammation is gonorrhœal in nature, and its complication is more common than blennorrhagic cervical catarrh. In very severe cases mixed infection plays an important rôle, since pathological investigation has shown that the duct is essentially the part affected. True abscesses are rarely formed. More frequently pseudo-abscesses develop—that is, a collection of pus in the already formed cavity of the gland. The inguinal glands are not affected in pure bartholinitis, since the lymph-channels of these glands pass upwards to the uterus. It is only when there is coincidently with bartholinitis involvement of the skin and mucous membrane that the inguinal glands are inflamed. As to the treatment, injections of a 1 per cent. silver nitrate solution into the gland ducts after evacuation of the pus—in cases of abscess by puncture or incision, in case of chronic inflammation by extirpation of the glands with closure of the space left

by buried sutures—offers the best prospect for cure.—*Monatshefte für praktische Dermatologie.*

Treatment of Adenoid Vegetations.—In a report upon this subject M. Helme gives his ideas upon the curative treatment of adenoid vegetations. The prophylaxis in persons of an hereditary lymphatic disposition, as well as in children who have had infectious diseases acting on the lymphatics, such as scarlatina, measles, etc., is without effect. The only cases in which it is useful are cases of acute coryza followed by adenitis, in which it is possible to prevent the progress of the affection by attention to the general health and to the local lesion. There is no prophylactic means of preventing the development of adenoid vegetations. Medical treatment is insufficient or useless in the great majority of cases. In reality there is but one method of treatment, and that is removal of the growths.

A careful examination should be practised in these cases, with the aid of anterior and posterior rhinoscopy and the index finger, carefully rendered aseptic. Having ascertained the situation, size, form, and consistence of the tumours, and the presence of pus in the naso-pharynx, it is necessary to determine whether there are any other obstructions to respiration, as rhinitis or hypertrophy of the pharyngeal tonsil. These complications have great importance as affecting the indications for operation. When such complications are present, there is an absolute indication to remove the vegetations and await the results. It is not common to see the permeability of the nose become re-established, and if the tonsils do not undergo regression, a second operation can easily be performed later on.

The contra-indications to removal of adenoid vegetations are but few. The first is haemophilia ; the second anomalies of the arteries in the naso-pharynx, which, according to Moure, are much more frequent than is supposed, and, finally, operation should not be undertaken during family epidemics of whooping-cough, measles, or influenza, nor in cases in which an acute or subacute catarrhal condition of the respiratory passages is present. Local inflammation of the diseased region is not an absolute contra-indication, but rather a cause for postponing the operation until all trace of mucus has been removed from the pharynx by means

of appropriate treatment. The day before operation injections into the nose should be avoided, and simple applications recommended, as resorcin and glycerine, one to fifty; insufflations of aristol and sugar of milk in equal parts, and the introduction into each nostril, night and morning, of boric acid ointment with or without menthol.

The operation varies in technique according as the patient is an infant, a child, or an adult. A child five to seven years of age should be left in ignorance as to the operation, and anaesthetized with bromide of ethyl, 5 to 10 grammes ($1\frac{1}{2}$ to $2\frac{1}{2}$ drachms) being sufficient. The greatest inconvenience of this anaesthetic is that it produces contraction of the jaws. By means of a mouth-opener the jaws can be slightly opened, a tongue-depressor pushed in until the contraction is over, and the operation can be done either with the curette or with the curette and forceps. The child should be placed on a plane a little higher than the operator, and the thorax should not be compressed by the clothing. The tongue-depressor being in position, the curette is introduced vertically into the cavity, with the extremity to one side; once in the cavity, the knife is turned so as to become horizontal. At this moment the curette is carried to the top of the vault, then drawn forward as if to press against the choanae. This second stage, upon which Moritz Schmidt rightly insists, is of capital importance, as it enables one to include the retro-choanal vegetations. In a third stage the curette is lowered by means of a circular movement from above downward and from before backward. A free cut is made with the curette in the median line,—one to the left and one to the right. If necessary, five or six such cuts may be made. The degree of force necessary must be determined by the consistence of the tumours. There may be some difficulty in bringing out all the vegetations removed, and the use of the finger (rendered aseptic) may be necessary to ascertain if all the fragments have come away. If the forceps and curette are used, the former is first introduced, the teeth being horizontal until the pharynx is reached, when they are turned and carried to the vault of the palate, opened as far as possible, then closed, crushing everything that can be seized with them. They are then drawn out, giving a turn with the wrist so as to detach a larger portion of tissue than is included in the blades.

If necessary to use the forceps a second time, the cutting parts must be disinfected with carbolic acid 1 to 20. The operation is then finished with the curette as described above.

For nursing infants anaesthesia is not necessary, and Lubet's small, special forceps should be substituted for the curette. In adults insufflations of cocaine are sufficient, the head being held immobile by an assistant.

The Universal Medical Journal.

Material for Skin Grafting.—Dr. Lusk has found that the epidermis raised by vesication could be successfully employed in skin-grafting, the cutis being separated from the cuticle, thus forming an ideal material. He has described the case of a workman who had fallen into a pan of boiling brine, head foremost. In attempting to remove him he again fell into the pan, and when rescued it was found that more than two-thirds of the surface of his body had been burned. There were profound constitutional symptoms, and his condition was considered hopeless. A month after the accident, however, favourable symptoms appeared, and Dr. Lusk thought he might possibly get well, though he was extremely emaciated, and nearly one fourth of his body was a raw, granulating surface. As no one would consent to furnish strips of skin, it was impossible to utilize the Thiersch method. An idea was then suggested by the following condition: There were numerous patches of exfoliated epithelium, the remains of vesication. They were hard, dry, and crisp, having for nearly five weeks been separated from the cutis. Why could not this material be utilised for skin-grafting? Dr. Lusk had faith to believe that it could, and therefore made grafts, using the following material: Attached to the dorsum of the right foot by one edge, the other being free and raised one fourth inch, was a patch of dry, exfoliated epithelium, extending nearly across over the surface, near the distal end of the metatarsal bones; the floating part was from one to one and a half inches wide. From this he clipped off a piece one inch square, softened and sterilized it in warm boric-acid water, and divided it into twelve grafts, which were applied to the anterior granulating surface of left thigh. The result was eminently satisfactory. Seven out of the twelve grafts took, and rapidly developed into

vigorous islands of skin. The subsequent treatment consisted in using this dried epithelial tissue, with which these large raw surfaces were covered with substantial skin, five months after the accident.

The man began sitting up a few days prior to this date, and with the attendant's aid could walk about the room. A month later the following experiments were made in the presence of an assistant and others: A little to the right and two inches below the trochanter major of the left leg, there was a raw surface three and a half inches in diameter. At the proximal end of the great toe there was a dry, bleached patch of epithelium which had been thoroughly isolated from the body seven weeks, being held only by ends of hair. With this material this raw surface was completely healed in twenty-one days. The man recovered with perfect use of the joints and the entire absence of cicatricial contraction.

Dr. Lusk has also described a case in which he produced a blister and used the epithelium as a grafting material. The patient was a woman, aged 52 years, who had a large varicose ulcer two and a half inches in diameter, three inches above the ankle on the outer surface of the left leg. She had worn elastic hose and tried all kinds of ointments for eight years. The granulations were unhealthy and bathed with a foul-smelling discharge. The treatment consisted first in thorough curettage, followed by stimulating applications, so that in ten days the granulations appeared healthy. A surface on the left thigh near the anterior superior spinous process was made aseptic, and on it was applied a piece of emplastrum cantharides two inches long by one inch wide (first moistened with carbolized oil). Vesication was produced in six hours, when the plaster was carefully removed. The epithelium was detached at the edges of the blister, washed in boric-acid solution, after which all moisture was absorbed with sterilized cotton, and it was suspended in a four-ounce salt mouth-bottle (aseptic cotton being used for a stopper), and kept at a temperature between 55° and 70° F. (12.8° and 21.1° C.). It was thoroughly dry in three days, when a piece one inch square was divided, making twelve grafts, which were applied in the usual way. The results were extremely gratifying. Nine of the twelve grafts took nicely and grew rapidly, so that in one month this ulcerated surface was healed, having a substantial epithelial covering.

Medical Record.

Treatment of Snake-Poisoning.—In *The Indian Lancet* of 16th August, 1896, a correspondent writes:—A few words with regard to treatment. As in 90 per cent. of the cases the bite is either on the hand or foot, few persons can be in such a destitute condition as to be without the means of their own salvation, and that is a ligature promptly and properly applied. A pocket-handkerchief, necktie, or piece of the shirt, if tied tight enough so as to stop arterial circulation, is nearly always available and sufficient. Then scarify deeply and suck the wound.

With regard to sucking the wound.—In all the directions in books and those issued to the police and schools, the recommendation to suck the wound is accompanied with a fatal caution, viz. "If the mouth be free from wound or scratches." The caution is absolutely absurd, as anyone can readily test for himself on the back of his hand, and by considering the mechanism at work. When a part is sucked the pressure is equal all round, and results in the saliva of the mouth and the material from the part sucked collecting in the hollow of the bent-up tongue, from which it can be instantaneously ejected. It would be a mechanical impossibility, even if the whole surface of the mouth were raw, for any material from the wound to find its way into the tissues during the act of suction. So that the directions should read—"Suck the bite vigorously on every occasion, whether the mouth have scratches or not, as no possible harm can ensue."

I am thoroughly convinced that if a ligature be properly applied within four minutes (and possibly much longer) of receiving a bite, and the part scarified deeply and vigorously sucked, no symptoms dangerous to life would follow.

I have seen several cases where a complete bite has been given by a truly poisonous snake, and where the above measures had been promptly applied, no symptoms of poison following.

A point on which I would like to lay great stress is the necessity of ascertaining the nature of the bite before beginning the injection of strychnia hypodermically. There is a widespread tendency to its indiscriminate use whenever a supposition of snake-bite arises. Persons really under the influence of snake-venom are exceedingly tolerant of strychnine, and hence in such cases large and repeated doses have been advocated. But should it transpire that the case dealt with be one of pseudo-poisoning, and the same large doses administered, dangerous or even fatal results might follow. As to the action of strychnia in snake-bite, my experience is that it is useless where a lethal dose of poison has been received. In such cases the only result I have seen is strengthening of the pulse. I have noticed nothing in the use of strychnia that could not be brought about equally well by other less dangerous stimulants.

THE CLINICAL JOURNAL.

WEDNESDAY, SEPTEMBER 23, 1896.

A POST-GRADUATE LECTURE

ON

SOME METHODS OF PERFORMING INTESTINAL ANASTOMOSIS.

Delivered at the West London Hospital

By LEONARD A. BIDWELL, F.R.C.S.,

Senior Assistant Surgeon to the Hospital.

OPERATIONS for re-establishing the continuity of the intestine after resection, although of great interest to the whole of the profession, are rarely seen by any but an operating surgeon and his assistants, since the necessity for their performance usually occurs more or less unexpectedly in the course of an operation. The chief conditions in which resection, or suture, is required are: (1) Gangrenous bowel in strangulated hernia; (2) Laceration of gut while separating extensive adhesions; (3) Removal of new growths of the intestinal wall; and (4) in the cure of an artificial anus. As we are never certain when such an emergency may arise, we have very little opportunity of asking you to see these operations, so I have thought that it might interest you to see some of the various methods performed on these specimens.

The first appliance necessary in any resection of intestine is an efficient clamp to prevent any escape of the intestinal contents during the operation. Without doubt the best clamps are the fingers of a good assistant, since the gut is less liable to be injured by the simple pressure of a finger than by a hard instrument; this form of clamp, however, necessitates an additional assistant, and is therefore often impracticable. Next to the fingers of an assistant I would suggest Maunsell's method of clamping the gut with a safety-pin and a piece of sponge. The pin is passed through the mesentery, then through one end of the sponge, and finally, after being passed through the other end of the sponge, it is fastened, and the gut is compressed between the pin and the

VOL. VIII. No. 22.

sponge. I will now draw your attention to Lane's clamp, which consists of a pointed steel rod with a cap at one end, and an india-rubber band: the rod is passed through the mesentery, and the gut is compressed between the rod and the band. Makins's clamp, one of the earliest, consists of a pair of spring forceps with long blades, covered with drainage-tube; the forceps are screwed up when in position. Since the mesentery is not transfixed there is a danger of the clamp slipping unless a considerable amount of pressure is exercised. Lastly, I will show you the commonest and one of the best ways of clamping the gut. In this method, a piece of small drainage-tube is passed through a hole in the mesentery, and either tied or secured with artery forceps over the gut; but here, I would caution you, it is absolutely unnecessary to tie the tubing tightly, since a very slight degree of pressure will occlude the lumen of the gut. There is, moreover, some danger in using artery forceps to fasten the tubing, since I have seen a distended intestine ruptured by the points of a pair of artery forceps which were controlling an elastic clamp.

There are three general points to be considered in deciding upon any method of joining a divided gut; they are—

1. That adequately broad and sufficiently wide surfaces of healthy intestine should be in contact.
2. That, although it is advisable to exclude the mucous membrane from the stitches, the fibres of the submucous coat must always be included.
3. And that the operation be performed as rapidly as possible.

I might also add that when not absolutely impossible, the operation should be performed outside the abdominal cavity.

Before describing the various methods of performing an anastomosis, I ought to mention the best form of needle and best material for sutures.

With only one exception the straw needle is the best for all the various methods; its size should be either No. 8 or No. 6, and it should be used without a needle holder. These needles do not make such a gash in the walls of the gut as

is made by the ordinary surgical needles with sharp edges. The very worst form of needle is the Hagedorn ; this formidable weapon makes a suture tract through which faecal extravasation can readily occur. I prefer straight needles, since it is impossible to pick up the submucous coat with any certainty with a curved needle.

For sutures I do not think that any better material can be employed than fine Chinese twist silk which has been boiled for one hour in a (1 in 1000) solution of bichloride of mercury. Many surgeons use chromic catgut, but this substance, besides being difficult to sterilize, is uncertain in its strength ; in some cases it stretches after twenty-four hours, and so might allow an escape of intestinal contents. I need only refer to the other substances used in order to condemn them ; they are silkworm gut, silver wire, horse hair, &c.

The principal methods of performing anastomosis may be classified according as the cut ends are united by—

- (a) Sutures alone.
- (b) Mechanical means alone.
- (c) A combination of sutures and of mechanical means.

Under class (a) and (b) we must make another classification according as the anastomosis is end to end or lateral.

Under class (a) I will bring under your notice three principal methods : the first, the Czerny-Lembert, is the oldest of all ; the mucous membrane alone is united with a row of interrupted sutures, and the serous and muscular coats are then united with another row of sutures. I may mention that, though this method bears the name of surgeons who flourished in this century, a successful case was effected as long ago as 1730.

The second method is one recommended by Dr. Halsted, of the Johns Hopkins Hospital, and the principle involved in this stitch has made the greatest advance in intestinal surgery. Halsted's method, though designed for lateral approximation, owes its importance to the recommendation that every suture should include some fibres of the submucous coat. I will give his directions for passing the sutures in his own words. "Each stitch should include a bit of the submucosa, since a thread of this coat is much stronger than one of the entire thickness of the serosa and

muscularis. It is not difficult to familiarize oneself with the resistance furnished by the submucosa ; the needle should be introduced by pressing the blunt end with the pulp of one of the fingers, when it will be found easy to pick up a bit of this coat with each stitch." He describes his method of anastomosis as follows :—"Six square, or quilt sutures, are inserted in a row near to the mesenteric borders of the knuckles of intestine, and are tied. At each end of this posterior row of sutures, and nearer the convex border of the intestine, two lateral square stitches are inserted and tied ; a little beyond the convex border of the gut, the eight or nine stitches, which constitute the anterior row, and complete the oval, are passed, but not tied. They are then drawn aside to make room for the knife or scissors, with which the two approximated intestines are then opened. The sutures of this anterior row are finally tied under a constant gentle irrigation with tepid sterilized salt solution."

The last method of performing anastomosis by sutures alone is that devised by the late Dr. Maunsell, who describes it as the way in which a tailor sews a sleeve into a coat. It is performed in the following way :

An incision is made in the convex border of the gut, just below the part to be sutured, and two sutures are inserted, one uniting the mesenteric, the other the convex surfaces of the cut ends. These sutures are tied, and their ends dragged through the incision made in the convex border of the intestine. By pulling on these the lower part of the bowel is invaginated on the upper, and the cut ends are made to present through the incision in the gut. The cut ends are then united by interrupted sutures, which are passed through all the coats of the gut, and are tied, the knots being inside the gut ; they are placed about one-eighth of an inch apart, and the ends are cut short. The invagination is then reduced, and the opening in the gut, below the joint, is closed with three or four Halsted's stitches. Some surgeons have recommended that an additional row of sutures should be inserted outside the joint after the reduction of the invagination, but this was not considered necessary by Dr. Maunsell.

This method, which effects a very firm joint, is, however, objectionable, since the sutures pass through all the coats of the bowel, and so a tem-

porary faecal fistula may follow the operation, as occurred in three otherwise very successful cases in which the method was employed in this hospital. In all the cases the fistula did not appear till the tenth day, and then quickly disappeared.

Under heading No. 2 the most important appliance for end to end anastomosis is Murphy's button. The button itself is probably well known to you, since it is advertised in every medical paper. It is applied in the following way :

A continuous silk purse-string suture is passed through all the coats round the edge of the divided gut, and one half of the apparatus is inserted within the lumen, and the silk suture is tied tightly. After the other half of the instrument has been fixed into the other end of the gut in a similar way, the two halves are pressed tightly together, and the anastomosis is complete ; care must, however, be taken that the knots of the silk sutures do not prevent the halves of the apparatus from coming close together. No outside supporting sutures need be used.

This method is certainly the quickest to perform, and in the hands of its inventor gave almost phenomenal results. The results in this country have not been so good, especially when the apparatus has been used to unite intestine after resection during acute obstruction. In several cases, too, the button has produced ulceration and perforation of the intestine below an otherwise successful joint. According to Murphy, this accident is due to the use of a button of too large a size. In a case of acute obstruction, as the intestinal walls are congested at the time of resection, the cut ends will not be together after the congestion has subsided, unless the spring of the button acts efficiently ; the clogging of the spring with blood would prevent its acting, and give rise to faecal extravasation. To my mind, it is unsatisfactory to trust a patient's life to the efficiency of a spring, when we have methods which do not involve such a risk.

Senn's method is almost an operation of the past ; it affects a lateral and not an end to end anastomosis. The opening left seems to have an almost irresistible tendency to contract, especially if silk sutures be used, when they will be found to hang in the opening and collect food, and so further narrow the opening.

Its performance is readily effected in the

following way. Two loops of gut are clamped, and openings made into their lumina about midway between the mesenteric and convex borders. The four threads attached to each plate are then armed with needles, and one of the plates is then inserted into each opening made in the gut. The two pieces of gut are then brought together, and the threads of one plate are tied to the corresponding threads of the other plate ; this completes the joint.

Under the third heading I would draw special attention to Mayo Robson's bone bobbin, since this method has certainly been followed by most successful results. As you will see, the bobbin does not in any way join the two cut ends of intestine ; they are united entirely by sutures, but the bobbin has the following uses :—

- (1) It enables the surgeon to use a continuous instead of interrupted sutures.
- (2) It acts as a splint to the newly sutured gut, and keeps the parts at rest.
- (3) It maintains a free channel through the anastomosis.

It is applied in the following way. A fairly stout continuous suture is passed through all the coats of the gut, uniting the two ends together over the bobbin. Another continuous suture is then inserted beyond the first, which unites all the coats with the exception of the mucous membrane. The joint is then complete. A curved needle has to be used in this operation.

To Paul's tube we need not now refer in detail, since it is seldom employed except by its inventor. It is a method which produces an artificial intussusception. The decalcified bone tube is stitched inside the one end of the divided gut, and two long sutures are passed first through each side of the tube, next inside the other portion of the gut for about two inches, and are finally passed through the walls of this from within outwards ; the two ends of the gut are united with a continuous suture, and the two traction sutures first passed are then pulled upon, and produce an invagination of the portion of the gut into which is stitched the bone tube. A row of Lembert's sutures prevent the return of the invagination, and the operation is completed by cutting short the two traction threads. This method, however, appears to give rise to a good deal of subsequent cicatricial contraction.

A POST-GRADUATE LECTURE
ON
**SOME COMPLICATIONS OF MIDDLE
EAR SUPPURATION.**

Delivered at the London Throat Hospital, June 22, 1896,

By WILLIAM R. H. STEWART,
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GENTLEMEN.—I propose this evening to mention a few of the complications which may follow a discharge from the middle ear. Fortunately, nowadays, not only are medical men becoming better educated in special subjects, but the lay public are beginning to realise the grave danger that exists if a discharge from the middle ear is allowed to go on unchecked. We therefore do not now see so many cases as formerly in our consulting rooms, and it is rather among the hospital patients and those just above them in *status* that those complications are so frequently met with. These patients, either from ignorance, wilful neglect, or want of time to properly look after their children, take no notice of a discharge, however offensive, until something serious in the form of mastoid trouble supervenes. It is to this latter and to intra-cranial conditions that I wish especially to draw your attention to-night. Before doing so, however, I would briefly say a word about the treatment of the discharge itself, and the other complications, which are usually so serious from the fact that, if neglected, one or other of these intra-cranial or mastoid conditions follow in their train.

With regard to the discharge itself, I always lay down the following rules for treatment:—In the poorer class, where neglect and dirt assert themselves most, I have found that iodoform emulsion, composed of iodoform one part, rectified spirit q.s. to moisten, boiled distilled water two parts, glycerine seven parts (Martindale's formula), is a great success. It is essential, if possible, for the surgeon to gently and thoroughly cleanse the ear himself the first two days, and instil the emulsion; afterwards the patient or parent should do it at

home morning and evening. It is better, unless absolutely necessary, that the patient should not syringe the ear, as they usually do it very roughly, and do more harm than good by the irritation they create. The emulsion clings to the meatus and tympanic walls, and so keeps the cavity sweet. Where the smell of the iodoform constitutes an objection, loretin, an American powder which has no odour, is a very good substitute. If you can see your patient daily, and have time to thoroughly attend to him yourself, a good plan is to wash out carefully with an acidulated solution of mercuric perchloride, 1 in 3000, using an intra-tympanic syringe when necessary; then gently stuff the meatus with antiseptic wool, piece by piece. If you cannot see the patient regularly, and you have to trust to him, you may take the following as your guide:—If there is a simple discharge without foetor, use iodoform emulsion, or a boric acid lotion 1 in 20; or, if you prefer dry treatment, boric acid powder or loretin. If foetor be present, and is such as to indicate carious bone, sulphurous acid 1 in 8. If any granulations are present, boric acid gr. x in $\frac{3}{4}$ j. of rectified spirit. I generally prefer to start these latter lotions with equal parts of water, gradually increasing their strength until they can be borne quite strong; you will find that they can be more quickly borne at full strength by this means. If much inflammation is present, a lead lotion of about two grains of acetate of lead to the ounce should be used as hot as can be borne, mustard leaves applied over the mastoid, and, if necessary, leeches in front of the tragus. If there be only slight inflammation, the lead lotion, luke-warm, is sufficient. Never put on less than two leeches, generally four, and always be careful to previously insert some cotton wool in the meatus, or the leeches will get in there and do a lot of damage. I think that if you lay this down as a general rule, with whatever variations you like, you will be successful in your treatment.

With regard to most of the complications other than mastoid or intra-cranial ones, I would say that really common-sense treatment is all that is necessary. For the ulcerative and eczematous conditions of the lobule and concha, extreme cleanliness is the essential point; and if anything further is required, a boric acid ointment is usually sufficient, taking care that the scabs formed are well soaked off with carbolic oil before the oint-

ment is applied. Sometimes the deeper points of ulceration require touching with nitrate of silver to hasten the cure. The adhesions and cicatricial bands that sometimes form in the cavity of the tympanum, binding down the membrane more or less, are naturally very fatal to good hearing, and also very difficult to deal with. The more recent ones are sometimes broken down by the aid of Politzer's inflation and the pneumatic speculum, but you will be very fortunate if you are able to do this. If you fail, it is best, unless there is unbearable tinnitus, to leave matters alone; for you may divide the bands with a knife of the kind I show you, a not particularly easy performance, with the almost absolute certainty that they will reunite after division. Politzer's inflations should be freely employed to prevent the adhesions reforming. If, however, the tinnitus is unbearable, an effort must be made at any cost to the hearing power, to relieve the condition. With this purpose in view, Sexton's operation should be performed—that is, the membrane and ossicles should be removed entirely, with the exception of the stapes. This can be afterwards easily removed if the tinnitus is not relieved without it. The great objection to this operation, in my mind, is that, with very few exceptions, the hearing power is lost, the membrane being rapidly replaced with cicatricial tissue.

With regard to polypi, I believe Mr. Wilkin will devote his demonstration next week to this subject, therefore I will only impress upon you the imperative necessity of removing the growth as soon as perceived, for serious if not fatal consequences may follow the penning up of pus behind the growth; and remember that great perseverance and attention are necessary on the part of both the patient and his medical adviser to complete the cure *after* the removal of the growth. Nowadays, I am glad to say, medical men are beginning to thoroughly recognise the nature and dangers of the growths; but even at the present time we now and again come across a case such as I will mention, but these usually have been treated by the older school of men, who seem to think that specialists are their natural enemies, and obstinately refuse to take anything in the shape of advice or hints from them. The case I refer to only occurred the other day. A woman, aged 25, had a large polypus one and a half inches long tightly

wedged in the meatus, and projecting about a quarter of an inch beyond the opening, there being only a very fine channel, through which some very offensive pus oozed. She was suffering great pain in the side of the head, and an old scar behind the ear showed evidence of previous mastoid trouble. The patient got well after the growth had been removed, but with absolute loss of hearing on that side. She had been told by her medical attendant to on no account do anything to the ear, as it would get well by itself. I have not the slightest hesitation in saying that had the case been allowed to go on for a very short time longer, the patient's life would have been sacrificed.

Somewhat allied to polypi are the granulations found in the external and middle ear, for although the smaller ones have no distinct epithelial coat, the larger ones have, and are composed of round and oval cells contained in a hyaline stroma, and interlaced with blood-vessels. They grow from the membrane, the cavity of the tympanum, or the meatus, appearing as small, soft, round, red growths, varying in size from that of a pin's head to that of a pea, and they bleed freely when touched. Cleanliness, and some boric acid or loretin powder insufflated are frequently all that is necessary to get rid of them; but they may require scraping with a ring-knife or some cautery or caustic application, nitrate of silver and chromic acid being the two agents most frequently employed. Of course, if carious bone be present, it must be thoroughly scraped. Puncture of the granulation before applying the caustic will frequently hasten their disappearance. Hot water is the best haemostatic. Inflammation or irritation of the meatus or tympanic cavity may produce a thickening of the bony tissue of a meatus—a hyperostosis,—or a more localised and distinct new bony growth, such as an exostosis. In the former, if the thickening does not subside with the inflammation after the removal of all sources of irritation, careful painting with iodine may be tried. If this has no effect, and the lumen is not completely closed, I would strongly advise leaving the case alone. But if it is so closed as to render the penning up of pus in the tympanic cavity likely, then recourse must be had to the mallet and chisel, or dental drill. Exostoses are usually divided into three kinds: the spongy, the

ivory, and an intermediate variety, which partakes of some of the characteristics of the other two. The spongy is a softish pedunculated single tumour, of rather rapid growth, easily removable by the snare. The ivory form is broad-based, slow-growing, usually multiple, and is most commonly seen on the posterior wall. The growth is covered by a smooth white skin. The intermediate variety is harder than the spongy, but less dense than the ivory, which in other respects it resembles. As a rule, it is best to leave these tumours alone until they reach such a stage as to be dangerous by blocking the meatus, and rendering the patient absolutely deaf. If removal is decided upon, I would recommend the dental drill, used with or without turning forward the ear. It is not always necessary to do the latter, as they can frequently be got at through the natural channel. It is of course necessary to take the greatest care, when operating, to have plenty of assistance. The apices of the growths should be attacked, and of course an anaesthetic is necessary. Exostoses, in my experience, are extremely rare.

I now come to mastoid disease, which is a grave complication, owing to the ease with which the inflammatory mischief may spread to the important structures lying around, and so give rise to further serious intra-cranial complications. This disease may be divided into the superficial and the deep. In the former the periosteal covering, and in the latter the interior of the process, are the parts affected.

The superficial mastoid disease may be met with in connection with an acute attack of otitis media, or it may arise from a direct blow on the process; but more frequently it occurs in cases of chronic suppuration, a sudden chill setting up acute periostitis, the discharge at the time suddenly lessening or stopping altogether. The onset is marked by violent pain and great tenderness, with redness and swelling behind the ear. This comes on suddenly, and, rapidly extending, may reach all round the ear, over the side of the head, and down the neck, the whole swelling at times having a distinctly erysipelatous look. The position of the auricle is very characteristic, standing out straight from the side of the head. Constitutional disturbance is at times very great, rigors and high temperature frequently occurring. An early diagnosis is most essential, in order to prevent

the much-dreaded extension to the brain and surrounding parts.

The treatment, if the inflammation be slight, is free leeching over the mastoid, followed by hot fomentations, the patient to be kept quite quiet in bed; but if these slight symptoms are not relieved in twenty-four hours, or if the pain and inflammation are great from the first, a free incision about three inches long, and down to the bone, dividing the periosteum, should at once be made. It is astonishing at times how deep one seems to go, owing to the swelling of the tissues. The cut should be from below upwards, starting from the tip of the mastoid and carried up close behind the auricle. The bare bone usually present should be freely exposed, any caries well scraped with a sharp spoon, and the surface freely doused with a warm antiseptic lotion to thoroughly wash away any débris. The incision must be kept open until any dead bone present has come away and the wound is thoroughly healthy. Of course, constitutional treatment must not be neglected.

In the deep form of mastoid disease—that is, when it is in the interior of the bone—the case is much more serious. It arises from pent-up pus in the cells, or is due to an inflammatory extension from the tympanic cavity. The chief symptom is the deep-seated and severe character of the pain, unrelieved by leeching, and the very slight amount of tenderness, with little or no swelling behind the auricle; though at times these latter symptoms are as bad as in the superficial form of the disease, and the two varieties may be combined. I wanted to show you an interesting specimen of mastoid disease, illustrating how large an amount of mischief may occur before the disease kills, but have not been able to lay my hand on the specimen. The girl had been attending one of our largest general hospitals, and her face had been regularly galvanised, &c., for facial paralysis. The foul abscess behind the ear had never been noticed, I suppose, as no treatment had been employed. When she came under my care she was in an extremely critical state, and she died shortly after the operation. P.M. showed a hole leading into the posterior fossa of the skull through the lateral sulcus, big enough to pass your thumb through; this was more or less blocked by a large mass, including thickened membranes and a thrombosed sinus.

In treating the disease, if it does not yield quickly to counter-irritation, hot lead fomentations, and saline purges, the mastoid antrum should at once be opened; and I cannot too strongly impress upon you the absolute necessity of early operation in these cases, and also in those of chronic middle ear suppuration that resist treatment and show no inclination to improve.

The indications for operating are—

- (1) When there is a suppurative catarrh of the middle ear, with persistent pain, unsubdued by incision or other means.
- (2) When suppuration exists in the mastoid cells, with no escape for the pus.
- (3) When there is suppuration combined with inflammation of the mastoid cells, during which vertigo and headache are developed.
- (4) When the suppuration resists treatment and the discharge is not great, but offensive.

The manner of operating is as follows:—Make an incision at least three inches long, and as close behind the auricle as possible; certainly not further away than half an inch, unless you want to open the lateral sulcus at the same time as in those cases in which lateral sinus thrombosis is suspected. The incision should be carried down to the bone, and the periosteum peeled back; then when any vessels that may spurt have been secured, a gouge, trephine, or dental drill should be applied. There is a small triangular space at the upper and posterior border of the bony meatus; this is the exact spot where it is best to apply the gouge or dental drill, and the trephine should be so placed as to include this triangle. In most cases I prefer to remove the posterior wall of the meatus as well. The instrument used should always be worked in a slightly forward direction, care being taken to examine with a probe from time to time as one goes along. In some cases the bone is softened and carious, and in these it is only necessary to use a sharp spoon and thoroughly scrape away all dead bone, which frequently extends over the side of the skull. Any granulation tissue that may exist must be scraped away, and sometimes the inspissated pus itself requires the scoop. In doing this care must be taken not to injure the facial nerve, as it passes through the Fallopian aqueduct, though if the disease be extensive this not infrequently takes place. The wound should be thoroughly washed out, and if

the disease is of long standing, thoroughly swabbed out with pure carbolic acid, a drainage-tube then inserted, and a dressing of antiseptic gauze applied. The wound should be dressed daily, and kept open until the bone is in a healthy condition. In those cases which come under heading No. 4, especially in young children, you are sometimes able, by breaking down the posterior wall of the meatus, to remove the collection of pus without perforating the outside bone. This will save the outer shell, and is certainly an advantage. Do not forget that it is possible to find the cells completely ossified.

Not only may the mastoid process be attacked by caries, but the ossicles and various other parts of the temporal bone, such as the roof of the tympanic cavity, the posterior wall of the external meatus, the plate of bone separating the mastoid cells from the lateral sinus, and the petrous portion containing the labyrinth, may also become the seat of disease. When the mischief is deep-seated, pain, though not always present, is frequently very great, and an abundance of granulations are thrown out when the tympanic walls are affected. The dead bone is often thrown off by an effort of nature, and in rare cases nearly the whole of the temporal bone has come away in this manner without a fatal result, and a few cases are on record where the labyrinth has come away entire. The large blood-vessels, such as the carotid and jugular, may be eroded, and facial paralysis may ensue from the portio dura being injured by caries of the Fallopian aqueduct. This is generally permanent. No life should be considered good by an insurance company where caries exists, for even if only the ossicles are involved, there is an element of danger from the liability of extension. The disease may, too, occur without a perforation being present. In giving your prognosis, however, you must to a certain extent be influenced by circumstances, more especially age and situation. Young children recover from mischief that could not fail to prove fatal in adults. As regards situation, disease of the tympanic walls is naturally much more serious than of the mastoid process. Fatal haemorrhage, pyæmia, phlebitis, meningitis, cerebral or cerebellar abscess, &c., are to be feared in any case of caries; haemorrhage from the carotid being, according to Roosa, especially apt to occur when

the disease is complicated with phthisis ; and this latter disease may itself follow caries, the morbid matter passing down the Eustachian tubes from the tympanic cavity, and so infecting the lungs. (McEwen).

The treatment should consist in removing as much as possible of the carious bone with the sharp spoon ; if out of reach, absorption may be attempted with a sulphurous acid lotion (1 in 8). Leeches and opiates must be used to relieve pain, and general constitutional treatment employed.

Until recent years all intra-cranial complications, as a rule, proved fatal ; but now, although much still remains to be done, the advance made in the surgery of these regions has robbed them of more than half their terrors. Abscesses are localised and evacuated, and the lateral sinus opened and washed out. If in a case of ordinary suppuration, more especially if a discharge from the ear has suddenly stopped, there comes on a feeling of malaise, with backache, nausea and vomiting, with or without a rigor, a dull aching pain over the mastoid region, which may radiate over the side of the head and down the neck, a foul tongue, frequent pulse, high temperature, and occasionally an attack of diarrhoea, with localised headache, you may be sure that some septic complication has taken place ; the localised and dull headache, as a rule, pointing to cerebral or cerebellar abscess, the more diffused and sharper one to meningitis. The pain, however, does not always localise the abscess, for in some instances of cerebellar abscess there may be frontal headache ; while, on the other hand, in a temporo-sphenoidal one the pain may be over the occiput. You may thus sometimes find very vague symptoms. But if with these cases you can get no definite symptoms, yet feel sure that some intra cranial trouble is present, I would most strongly urge upon you the necessity of operating without delay. As an instance of this I hoped to show you a boy whom I operated on three or four months ago, but he has joined a school treat, and will not be back in time. The notes of his case I will read presently. As a further instance of the unreliability of symptoms I would mention the case of a woman I recently operated upon for frontal sinus disease, who showed all the symptoms of that trouble. Polypi ran round the anterior portion of the middle turbinated, and a thick creamy discharge escaped when the

patient stooped forward ; in fact, I felt no doubt about where the exudation came from. I therefore trephined over the frontal sinus, and found the bone so absolutely sclerosed that there was no frontal sinus on that side, and the bone was nearly half an inch thick. When I came down upon the brain I found there was absolutely no pulsation in it. There was a history of pain on the side of the head for a period of two years. On opening the membranes there was no abnormal gush of serous fluid, and on probing the brain in various directions I could find no tumour. No optic neuritis was present at that time, but it has since developed rapidly, the veins being very full, and haemorrhagic spots were visible. I have had the patient very carefully examined, but no cranial nerves were implicated. Further operative procedure is refused. Still there must be a further operation at some time, and I cannot help thinking that we shall find a tumour of slow growth in the frontal lobe. The case is interesting on account of the absolute symptoms of frontal disease, though there is no frontal sinus on that side, the history of two years' pain, and the absence of any pulsation in the brain whatever. After incising the membranes there was still no pulsation, so that there must be pressure somewhere. I also saw a case operated upon a little while ago in which, with the exception of the straining backwards of the head, symptoms of cerebellar abscess were present, the cerebellum was explored without result, and P.M. a large tumour in the frontal lobe was found.

These brain complications may come on suddenly in cases where the suppuration is, to all appearances, yielding to treatment, sitting in a cold draught, or receiving a blow on the head being the exciting cause. The symptoms are at times very insidious, increased pain being rapidly followed by paralysis, coma, and death. Körner, of Frankfort, draws attention to the fact that serious intra-cranial mischief is more likely to follow disease on the right side than on the left, and the explanation he gives of this is that the right lateral sinus lies deeper and more forward than the left, the intervening layer of bone between it and the ear being thinner and more easily perforated.

Cerebral abscess more frequently occurs as the result of middle ear disease than from any other cause, and, according to Toynbee, follows more especially disease of the tympanic walls. It may

be acute or chronic, a diffused softening without any limiting envelope, or a well-defined abscess with a distinct membrane; it may be single or multiple, but the latter condition is rare. It is usually located in the temporal lobe, behind a vertical line drawn through a spot just anterior to the tragus, sometimes extending backwards into the occipital lobe, but seldom forwards. The pus may be superficial, and in direct communication with the diseased bone, or deep-seated, and separated from the bone by a layer of healthy brain substance; in the latter case the infecting organisms find their way through the veins, the arteries, or the lymphatics. The pus contained in the abscess may be either inodorous or intensely fetid, of a thick greenish consistency, or pale, thin, and scarcely purulent. The pain is dull and localised, with a tendency to greatly increase and become diffused, though, as I have just mentioned, the locality of the pain may be misleading. The temperature, at first high, is followed by a marked and steady fall to below normal, and does not rise again until the abscess is emptied, when it generally goes to normal. The pulse becomes slow, regular, and full; the respiration slow, shallow, but regular. The bowels are confined. The mental power is affected with slow and sluggish cerebration, lethargy, and drowsiness; there is a failure of memory, aphasia, and some paresis of the face. These conditions speedily disappear when the abscess has been thoroughly emptied; but if this stupor rapidly passes into coma the fatal result quickly follows, at times in forty-eight hours.

Optic neuritis is not constant, but is more frequent in abscess than in any of the other complications. (McEwen). Paralysis of motion and sensation, and convulsive movements may or may not occur, but if they do they are very useful in localising the seat of the abscess. Rigors, delirium, and irritability are also not constant symptoms. There is usually a muddiness of the skin and emaciation, the latter and the foul breath so often present in these cases being no doubt due to a stoppage of the proper digestive process (Barker).

With regard to treatment, the presence of an abscess being strongly suspected, an endeavour should at once be made to reach the pus without delay, for these cases never recover unless operated

on. Having turned down a large semicircular flap of all the tissues covering the selected spot, an inch trephine should be applied at a point two inches above Reid's base line—a line that runs from the occiput through the centre of the external meatus to the lower angle of the orbit—and one and a quarter inches behind the centre of the external meatus, and a crown of bone removed. The membranes being then incised, a fine trocar or director should be thrust into the brain substance in different directions. If pus be found the abscess should be freely opened, and the cavity thoroughly irrigated with a warm boric solution, a drainage-tube (glass preferred) inserted and brought out through a hole made in the flap, the flap itself properly adjusted, and the wound dressed with a light antiseptic gauze dressing. The abscess cavity should be gently washed out daily until healing takes place. The antrum also should be opened, all dead bone and pus removed, and a free drainage established. Unless the mischief be localised in the cerebellum, I now generally perform a modification of Dean's operation. I use a fairly large trephine, and open the lateral sinus and antrum at the same time. You can thus at once see if the sinus is infected, and by clipping upward with bone forceps can tap the temporo-sphenoidal lobe. When the abscess is situated in the cerebellar region I like to make a separate opening. I would here draw your attention to the difficulty there is in keeping in the drainage-tube in cases of abscesses in the brain substance. As soon as the contents are evacuated, the sides close up immediately, and show a strong tendency to push out the tube, which frequently is found next day lying in the dressing. For this reason I have lately employed glass drainage tubes, which keep in much better.

A cerebellar abscess has the same characteristics as to diffusion or limitation, contents of abscess, &c., as one situated in the cerebrum. The usual seat of cerebellar abscess is the anterior portion of one of the lateral hemispheres, where it lies in contact with the posterior surface of the petrous bone and lateral sinus, and, according to Toynbee, is most frequently produced by disease of the mastoid process, external meatus, or petrous bone. As a rule, the symptoms are much the same as in a case of cerebral abscess. The pain is dull and localised; the temperature is high at first, falls

rapidly to subnormal, at which it remains steady until the abscess is evacuated, when it rises to normal. The pulse is regular, slow, and full ; the respiration slow, shallow, and regular ; the bowels confined, and there is the same muddy look about the skin. There is, however, one symptom which is nearly always present, and which is pathognomonic of cerebellar trouble, namely, the straining backward of the head. About three years ago I had a case in which the occiput was touching the spine, and intense pain was caused when the head was moved. I trephined over the cerebellum, and evacuated about two teaspoonfuls of pus from just under the dura mater, formed by a circumscribed meningitis. The boy did well ; I saw him about a year ago, and he was then quite well.

An abscess in this situation, however, may exist without any brain symptoms at all until pressure is exerted on the peduncles. This is mentioned by Gowers in his book on the brain, and was very well illustrated by a case of my own, which I published in the *Lancet* in 1888. It was that of a boy who had suffered from a chronic suppuration, and was admitted under me at the Great Northern Central Hospital with extensive mastoid disease. The antrum was freely opened up and free drainage established. He seemed to be doing very well, was cheerful, free from pain, and able to sit up and freely move about the bed until an hour before death, when he suddenly became faint, cold, and collapsed, with an irregular pulse and jerky breathing, but there was no twitching nor paralysis of any kind. The post-mortem examination revealed a large abscess occupying the whole of the right hemisphere of the cerebellum. There was no optic neuritis nor straining back of the head,—in fact, nothing to point to brain mischief, although the whole of one hemisphere was one big abscess. There are also on record some cases where these abscesses have worked their way to the surface with favourable results.

The treatment is the same as for cerebral abscess—as early an operation as possible. The situation chosen for the trephine should be one inch below the base line, and two inches behind the mental centre. To start with, it is best to use a small half-inch trephine, the opening being enlarged by bone forceps.

Without doubt the most fatal of all intra-cranial complications is meningitis. It may be secondary

to some other complication, such as a cerebral abscess reaching the surface, thrombosis of the lateral sinus, or a dural abscess, or it may be uncomplicated, and occur within a few days of the commencement of the discharge from the ear. Should an intra-cranial complication set in very early in the course of the disease, it may safely be put down to meningitis. It may be divided into two varieties, the limited and the diffuse ; these being, it is said, due to two different micro-organisms. These organisms may be conveyed either through the blood-vessels and lymphatics, or they may find their way through the internal auditory canal from the labyrinth. The form which has a tendency to limit itself by plastic exudation is generally found over the roof of the tympanum, under the squamous portion of the temporal bone, or the lateral sulcus. This variety naturally gives the best results from treatment. The other form, the diffuse, shows no tendency to limit itself, but spreads rapidly, preferring the base of the brain to the vertex, and sometimes attacking the spinal membranes. The onset is sudden, and the course rapid. Pain is, as a rule, sharp and all over the head, and even where the mischief is localised the pain may be diffused, while in some cases there is no pain at all. The temperature, in the diffused variety, is very high ; in the localised not quite so high, but in both cases it is steady. The pulse is small, rapid, and irregular. The respiration is increased and irregular, and the bowels are confined. There may be listlessness, drowsiness and coma, or rigors, delirium, extreme restlessness, with convulsions, tremors and twitchings of the limbs ; hemiplegia, facial paralysis, convergent strabismus, dysphagia, aphasia, agraphia, and amnesia, emaciation and retraction of head. Optic neuritis is not generally present, for in most cases death occurs before it has time to develop.

I was called down to Maidstone to a consultation regarding a very sad instance of this kind two months ago. The child, a twin, had caught cold. She had been in a very excited state from helping at some bazaar, and acute inflammation of the middle ear had set in. The patient got rapidly worse, and in two days I was sent for to see her. I found that basal meningitis was present, and in about three days she was dead.

With regard to treatment of the circumscribed

variety, more especially if pus has formed, and you are able to ascertain the extent and to localise the trouble, the trephine should be used, and an attempt made to reach the pus. When that is found the abscess cavity should be irrigated with a warm boric acid wash, and drained. In the diffused variety no treatment is of any avail.

A very important complication is that of thrombosis of the lateral sinus, which may occur directly from necrosis of the posterior part of the petrous bone, but in most cases it spreads from the mastoid cells or posterior wall of the tympanum by the veins emptying into the sinus. The thrombosis may spread backward to the torcular herophili and downwards into the jugular vein. The clot formed may be of two varieties,—tough, with a tendency to organise and obliterate the vessels, and thus comparatively harmless; or, soft and friable, more septic in character, and, being loosely attached to the sides of the sinus, is very liable to break away, and, being washed into different parts of the body, may produce abscesses in liver, lungs, or kidney, or cause paralysis. This, of course, is the more serious form, and is evidently produced by a different organism from the tough variety. When death supervenes in these cases, which it usually does in about fourteen to twenty-one days, it is most frequently from pulmonary pyæmia, but it may be due to meningitis, general pyæmia, or an abscess on the brain. The onset of the disease is very sudden, but there is generally a history of a chronic mid-ear suppuration lasting a long time. Pain is very acute and lasting, and when situated in the posterior triangle and running downwards in the line of the jugular is a very valuable diagnostic sign (McEwen). Pain, too, is generally felt in the affected ear, there is a good deal of tenderness on firm pressure being applied to the posterior border of the mastoid, and below the occipital protuberance. Some œdema in the same localities may also be present. Stiffness in the muscles of the back and sides of the head is sometimes complained of. There is vomiting repeated day by day. The temperature is high; pulse and respiration increased in frequency. There is diarrhoea and giddiness, and delirium or convulsions may be present. Rigors take place, and the skin has a peculiar goose-skin appearance (Barker).

For treatment I now usually employ the opera-

tion I mentioned a short time ago—that is, I use a fairly large trephine to open the antrum and lateral sulcus at the same time. The sinus can more freely be laid bare by chipping away with the bone forceps. I then generally tap the sinus and examine the clot. If pus be present, and the clot soft and friable, then I cut down and tie the jugular in the neck. The vein is sometimes rather difficult to find, as it is so attenuated. Then returning to the sinus, it should be thoroughly slit up, and the clot scraped and washed away. A plug of antiseptic wax or carbolic gauze should be used, if necessary, to stop haemorrhage, all dead bone being of course removed where possible. If the clot is healthy and firmly fixed in the sinus it is as well to leave it alone.

I will now read you short notes of the case I mentioned a short time ago as having unusual symptoms.

A boy, æt. 11, had scarlet fever when four years old, and had had discharge from the ear ever since. Five days before admission he complained of ear-ache, was shivering, and seemed ill. The next day the pain and shivering continued, and he vomited. Two days afterwards the discharge from the ear suddenly stopped, he was delirious off and on, and had no sleep. He was admitted on February 26th, looking very ill, with great pain in the head; the discharge had been re-established. Temperature 97; pulse 120, regular. There was a good deal of swelling and tenderness behind the ear, some slight swelling of right disc; veins full. I at once opened the antrum and established communication with the middle ear. A good deal of pus came from around the tip of the process. Next day he was better; temperature normal, pulse 100, and regular; the swelling around the wound had subsided, and he took his food well. Some very offensive pus was syringed out of the antrum. The next day he became restless, had a good deal of pain, and the wound looked very foul and sloughy. The optic neuritis had greatly increased. I therefore, having first thoroughly cleansed the wound, trephined over the sulcus and the antrum wound; the sinus was tapped, and a firm, healthy clot was found. I then chipped away the bone upwards, removing a fair amount of it; the membranes bulged into the opening, and no pulsation could be felt. On incising the membranes a large amount

of serous fluid escaped. A director was thrust into the temporo-sphenoidal lobe in every direction, but no pus was found. The wound was dressed with blue gauze. He had a rigor the same evening, the temperature going to $104^{\circ}4$, but he passed a good night, had no pain, and next day I found him sitting up in bed eating his food, and much improved in every way. That evening he had another rigor, when the temperature rose to $104^{\circ}2$. As it continued to reach 104° every day, on March 2nd I again opened up the wound, thoroughly scraped out the clot in the sinus, explored the temporo-sphenoidal lobe again, also trephined over the cerebellum, but no pus could be found. The wound healed remarkably well, but for nearly a month, although the wound remained well and the boy seemed to be improving, he had a rigor nearly every day, with a temperature over 104° . On March 25th he complained of the other ear, and a small polypus was removed from it. The optic neuritis did not disappear, and the temperature rose to nearly 104° at least once a day. On April 6th, as the wound was healed, I told the house surgeon to get the patient up and let him run about. The temperature never rose after, and he is now well with the exception of some slight discharge from the ear.

Pyæmia is the last complication to which I wish to direct your attention. This may arise during any of the foregoing intra-cranial conditions, or may be entirely extra-cranial, arising from a simple middle ear suppuration, *fœtor* not being necessary for its production. It has all the characteristic symptoms of pyæmia starting in any other region of the body—such as a temperature varying from extreme height to subnormal, occurring at any hour, and having no connection with the formation of or discharge of pus; increased respiration and pulse, sweatings, with the characteristic smell about the skin, recurring rigors, and frequent diarrhoea.

All treatment has proved so very unsatisfactory that there is not much to be said about it. The great thing to be relied upon is, I am sure, abundance of fresh air, and keeping such sources as the middle ear antiseptic by frequent irrigation, as well as opening and treating antiseptically all secondary abscesses that may form. Quinine may do good in large and repeated doses. [Some patients suffering from the complications were then introduced and demonstrated by the lecturer.]

THERAPEUTICAL NOTES, &c.

Diagnosis of Calculus by the New Photography.—Dr. D'Arsonval's opinion is that a great diagnostic advantage will soon be reaped, in regard to renal and vesical calculi, by the Roentgen photography. The latest pictures obtained by Chappuis and Chanel have an especial value, since they show that it will soon be possible to diagnose calculi in the urinary passages with absolute exactness. They not only show the existence of a calculus in the bladder, kidney, or ureter, but it is possible to distinguish the substances of which it is composed, whether it is homogeneous or formed of different layers, whether the kernel is small or large, and of what it is composed. The most interesting photograph from this point of view showed—1, the silhouette cast by a calculus of pure uric acid; 2, that of a calculus the same size as the first, but composed exclusively of phosphate of ammonia and magnesia; 3, that of a calculus much larger than the others, formed of several distinct layers of uric acid in the centre, with an outer layer 4 mm. thick, and entirely different in colour, and composed exclusively of the triple phosphate; 4, the silhouette of a bone 1 cm. thick, and another of the index finger of one of the experimenters. The differences in the depth of shadow in this photograph are so marked that it is impossible to mistake the characteristics and kinds of the calculi. The tiny kernel of uric acid is distinctly visible, while the outer layers of the large calculus are represented by clearly defined rings. A second photograph showed another calculus with a kernel formed of soda urate, enclosed in an outer layer of the triple phosphate, both very clearly defined in the photograph. A third represented a number of uric acid calculi lodged in the parenchyma of the kidney, one-half of which was 5 cm. thick. The rays passed through this thick layer of tough tissue, and the calculi alone showed in the photograph. It will be a simple matter, therefore, preliminary to an operation, to take the photograph of similar calculi, and then compare them with the results of photographs taken through the patient.—*Bulletin de l'Académie de Médecine.*

DR. HALSTEAD, of Chicago, in a communication

entitled "Metatarsalgia, with a Report of three cases of what is termed Morton's Painful Affection of the Feet," concludes as follows—(1) That what is known as metatarsalgia is not in the beginning a distinct pathologic entity, but rather an early symptom of static flat-foot. In cases of long standing, irritation of the plantar nerves by pressure from flattening of the transverse metatarsal arch may cause an inflammation of the nerve, or even in some cases the development of neuro-fibroma. (2) That most of these cases can be permanently cured by following the treatment usually employed in beginning flat-foot, *e. g.* systematic massage, gymnastics, and the use of a properly fitted shoe, and in some cases application of a metallic brace to the sole of the foot. (3) In cases of long standing, where there is well-marked pathologic change in one or more of the branches of the plantar nerves, resection of the nerve should be performed; the more radical operation, such as resection of the metatarso-phalangeal joint or amputation of the toe, are not indicated.—*Medicine.*

The Use of Nux Vomica or Strychnine in the Diseases of Children.—In a recent article, Comby, after some preliminary remarks upon nux vomica and its alkaloids, takes up the indications and contra-indications for its use. He insists upon its great value in the treatment of the later stages of cerebral paralysis and paralysis resulting from lead-poisoning, in incontinence of urine from atony of the sphincter, and in all forms of toxic paralysis as represented by diphtheria. He also believes it to be a valuable remedy in spermatorrhœa, in hysterical paralysis both motor and sensory, such as aphonia, hemianæsthesia and amaurosis, and facial paralysis. He quotes Rousseau as stating that even in chorea full doses of nux vomica may be given with advantage. Strychnine is also useful in conditions of heart disease in which there is feebleness of this organ resulting from sudden collapse, or in the course of infectious disease. He also thinks it of value in the paralysis of embolism, in atony and dilatation of the stomach, and in constipation.

The only contra-indications are those in which there is marked reflex excitability of the nervous system.

After stating that the drug may be well administered to children in the form of small pilules or

cachets, he adds that he is in the habit of using small cachets containing the following powder:

B.	Powdered nux vomica	... gr. $\frac{1}{4}$
	Ciccarbonate of sodium	... gr. iiij
	Calcined magnesia	... gr. iiij
	Pepsin	... gr. ij

One such cachet is to be given morning and night, after meals, to a child of from three to five years; this treatment should be continued for ten days, then suspended for ten days, and then recommenced. The pepsin renders the stomach more tolerant, and is valuable for digestive purposes, while the calcined magnesia exercises a favourable influence over any tendency to constipation. If there is a tendency to diarrhoea, he replaces the calcined magnesia by salicylate of bismuth.

For atonic dyspepsia the following prescription may be used:

B.	Tincture nux vomica	... m. xv
	Tincture calumba	... 3ss
	Tincture gentian	... 3ss

Five to ten drops of this may be given after each meal to a child of five to ten years.

A prescription for incontinence of urine which Comby believes to be useful is in liquid form, as follows:

B.	Tincture nux vomica...	... m.v
	Tincture rhus aromatica	... m.xlv
	Tincture cinchona	... m.xlv

Ten to twenty drops at night before retiring.

Comby also believes that strychnine may be used by inunction, as follows:

B.	Lanolin or lard...	... 3j
	Strychnine	... gr. v

Rub a small piece of this ointment into the skin night and morning.

Should the child seem at any time during the administration of strychnine to be suffering from the excessive effects of the drug, the stomach is to be washed out, and narcotics such as choral and bromide of potassium, with inhalations of chloroform or ether, resorted to.

Senile Pruritus.—The 'New York Polyclinic' for July has an original article by Ellice Alger on the management of senile pruritus, which usually receives very little attention.

It rarely begins before sixty, generally originating as a temporary pruritus easily relieved, but gradually getting more persistent and extensive.

There is no visible primary cutaneous lesion. The secondary lesions assume the form of scratches and excoriations, and occasionally a dermatitis results. The itching is often worse at night, and is increased by change of temperature and clothing.

The whole skin is much thinner than normal, owing to absorption of fat and corresponding atrophy of other cutaneous elements. It is dry, bloodless, and poorly nourished. Exfoliation is delayed, and the outer surface is rough and thick and dry; the muscles atrophy and the skin becomes loose and folded, and considerable pigment is deposited. The secondary lesions, while almost always present, are not at all in proportion to the amount of violence done to the skin, and marked secondary lesions in a case of recent origin would be presumptive evidence of a mistake in diagnosis.

The prognosis should be very guarded, depending largely on the cause, if one be found. The heart is generally feeble, the arteries undergoing calcareous degeneration, and the changes in all the organs which follow arterial sclerosis are progressing. The kidneys are especially apt to be defective, and the tissues are loaded with waste products. The skin, which should lift the burden from the kidneys, is utterly unable to excrete owing to glandular atrophy, and many poisonous products are deposited in the skin as direct irritants to the peripheral nerve endings. The circulation in the blood of bile, sugar, and malarial organisms is also a factor in some cases.

There are some remedies that may prove beneficial in almost any case, such as cannabis indica, given in gradually increasing doses. Belladonna and gelsemium can be used in the same way as can valerian and the bromides. Certain cases are much benefited by the use hypodermically of pilocarpine, gr. $\frac{1}{10}$, and the alkaline diuretics are also very useful. For the dyspepsia and constipation nothing is more generally useful than equal parts of Tr. Nux Vomica and Ext. Cascara in sufficient doses. Diet should be regulated, avoiding the use of tea and coffee and alcoholics, and restricting the use of meats. Carbolic acid is one of the best antipruritics, Bronson's artificial oil being especially valuable.

B.	Liq. Potass.	3ij
Ac.	Carbol.	3iv
Ol.	Bergamot.	gt. x	
Ol.	Lini	ad	3ij
M. Sig.—Ext. use.					

Especially grateful at times are acid or alkaline baths, as are aqueous applications of acetanilid, one drachm to one ounce, which often gives relief for hours. Menthol and cocaine are much praised as antipruritics, but are of little value here.

Charlotte Medical Journal.

The Diagnosis of the Morphine Disease.

By J. B. MATTISON, M.D.

The purpose of this paper is to present certain facts along somatic lines that will clear away doubt in suspected cases. There are morphinists who, so far as outward symptoms under ordinary conditions obtain, present no proof. Again and again, in my experience, this fact has been noted; and if this be so with one whose professional life is exclusively given to the study of this disease, it goes without saying that it is much more likely to occur with one engaged in general work, by whom minor evidence of this toxic condition might easily be overlooked.

I have known a doctor to take morphine fifteen years and present himself for treatment. He was cured, and has been free six years, without showing the slightest sign of his drug disease. Of course, this was a very exceptional case, for usually the stamp of this neurosis is soon patent; but such a case is likely to re-occur, and possibly involve such interests along medico-legal lines as to make a correct diagnosis of more than common importance.

This case in point: Nine years ago the wife of a medical man brought suit against him for divorce. He was charged with being a morphinic. The charge was denied, and a countercharge made that she was an *habituée*. This was denied, and, in proof of denial, she was examined by two physicians, who gave evidence that she was free from the disease. They were mistaken—she had been taking morphine daily for six years! They failed to make crucial test of her true condition, and so erred.

Another case: In the appeal for a new trial for Carlyle Harris—who was killed for alleged murder of his wife with morphine—evidence was offered to prove that she was a morphinist, and so might have died from an overdose self taken. The appeal was denied, and in his opinion, refusing, Recorder Smyth—the trial justice—laid special stress on his belief that had Mrs. Harris been an *habituée* the fact would have been known to her

husband, in whose behalf on the trial no such claim had been made.

I have no hesitation in saying that in this part of his opinion Recorder Smyth made a grave judicial error. Why? Because many a case of morphinism in a wife has persisted for years unknown to her husband or even her doctor. That is a fact. Just such a case of morphino-cocainism is now under my care; and, granting that the judge's belief along this line was the main reason for his refusal, if this fact had been properly presented and insisted on by competent counsel, it might have secured a new trial for Harris; which was, in my opinion—the claim as to the morphine being undecided—undoubtedly his due.

Many and varied as are the tokens of this toxic neurosis, it is safe to say there is not a single symptom infallible as a sign of the disease. This statement may be contrary to the general opinion in and out of the profession, but it is true. Anything like a "snap-shot diagnosis" in morphinism may be quite unreliable, and should never be made. The usual various sequelæ—many of which may be present in other disorders—are known to you, and details need not detain. The point of most value in this paper is a statement of the fact that we have at command two tests that are certain to detect chronic morphine-taking. They are enforced abstinence and urinalysis.

Concerning the first, so imperative is the demand of the system for a sufficient supply of morphine at more or less regular intervals, when it becomes part and parcel of the daily need, that any withholding beyond a certain time is sure to be followed by symptoms that settle the narcotic status beyond doubt.

The length of this abstinence needful to determine the question varies according to temperament and condition; but as patients require the drug daily, or usually more often, forty-eight hours' withdrawal will suffice for proof. Possibly, in some extraordinary case, a longer time may be needed; but, as a rule, two days will do.

Regarding the renal test, various methods will serve; but the simplest of which I know is that of Dr. E. H. Bartley, professor of toxicology in the Long Island College Hospital. This is the Bartley process: To the suspected urine add carbonate of sodium to make it alkaline. In this put a portion of chloroform; shake well, allow it to settle, draw

off, and add a small amount of iodic acid. If morphine be present, a violet tinge appears.

With a consensus of symptoms usually noted, and the time and urine tests, the diagnosis of the morphine disease need never be long in doubt.

Medical Record.

Surgical Treatment of Carbuncle.—O'Conor states that during the past three years it has fallen to his lot to treat twelve cases of carbuncle—six on the nape of the neck, two on the buttock, two on the back, one on the perineum, and one on the face; in each instance the treatment adopted was excision, supplemented in four cases by erosion.

The operation is carried out in the following manner.

1. The patient having been placed under ether anaesthesia, a deep incision is made around the whole circumference of the carbuncle, at least half an inch outside the infected area. Luckily, carbuncles, as a rule, do not occupy parts where there are any large blood-vessels; therefore there can be no excuse for stinting the depth of this incision.

2. With the handle of a scalpel or a periosteal elevator, the carbuncular mass is undermined, elevated, and removed, in the same manner as a tumour of the breast.

3. Sometimes it happens that the disease has extended too deep, and cannot be isolated from the surrounding structures; in such a case, after all has been removed that can be with the knife, strong scissors are brought into action, and the business is finished with a sharp Volkmann's spoon. Not a trace of the disease should be left.

4. In order to contend with any microscopical particles that may have escaped this process of eradication, the whole surface is to be swabbed with a solution of zinc chloride, forty grains to the ounce. Of course, this is only used in cases that do not admit of a clean removal.

5. A large circular wound is generally left; this is painted with a solution of carbolic acid, alcohol, and methyl violet, one part of each, in water, ten parts. It is then dusted with iodoform, packed with iodoform gauze, and bandaged.

6. When the granulating wound comes level with the surrounding skin, Thiersch's grafts are applied, which considerably hasten the cure.

Needless to say, this operation is not by any means a bloodless one, but with a steady assistant armed with a dozen pressure forceps, there is no need for alarm. Any vessels that merit it are tied ; the general oozing soon ceases with the pressure of a sponge soaked in hot water.

Out of the twelve cases, in eight the carbuncle was cleanly excised ; the remaining four required the scissors, spoon, &c. In not a single instance did the disease recur in the wound ; constitutional symptoms, in some cases severe, vanished in twenty-four hours. In fact, it is hard to realise that a patient so deep in the throes of septicæmia can be restored to his normal state in such a short space of time. No other method that the author has ever tried or heard of equals complete excision in this respect.

O'Conor then details a case in which he practised this treatment successfully, and terminates the paper by saying that having obtained such remarkable results by excision, he is impelled to question the dictum of so good an authority as Mr. Jonathan Hutchinson, jun., in 'Treves' System of Surgery,' page 713. This gentleman, writing on the treatment of carbuncle, states : "An ancient plan of treatment of carbuncle has, however, been lately revived, consisting in cutting with scissors and scraping away all the slough in the belief that this shortens the healing process. Undoubtedly, some time and perhaps considerable pain are occasionally saved by this plan ; but, on the other hand, the risk of opening up the veins and causing pyæmia appears to be distinctly increased, and many surgeons have on this account abandoned it." O'Conor explains the occurrence of pyæmia as due to half-hearted operations where the disease was not thoroughly cleaned away, or to operations undertaken too late, when the pyæmic virus had already entered the blood. So far he has not heard of pyæmia following Barker's operation in psoas abscess, scraping away suppurating inguinal glands, or the opening up and scraping of large abscesses on the thigh, where the process is somewhat identical.

The grounds for his criticism are : First, that he does not think there is any scientific foundation for the remark just quoted as to the possible dangers ; secondly, he is a strong believer in the doctrine that, in every instance in surgery where there is a grave constitutional depression, prompt

measures ought to be taken, and, if possible, a "clean sweep" made, not only of the micro-organisms, but of their camp. To dilly-dally, in the hope that painting with iodine may eventually conquer, is a species of surgery that must be attended with disaster.

New York Medical Journal.

Chronic Otorrhœa permanently cured with Trichloracetic Acid.—Halasz confirms Okuneff's announcement of the value of this treatment, and recommends the following *modus operandi* : As the pain is severe, five to eight drops of a 10 per cent. solution of cocaine should be held in the ear for three minutes. During this time a syringe of tepid water should be used to melt the crystals of the acid on the specially constructed sound. The inner ear is then lighted and the sound introduced into the middle ear through a rubber ear speculum. Every spot to be cauterised should then be touched lightly but effectively with the acid, especially the edges of the perforation and the mucous membrane of the middle ear. The ear should then be rapidly rinsed out with one or two syringes of water, and after it is dry, dusted with aristol or powdered borax blown in. The operation should be rapid and repeated once or twice a week, when the otorrhœa soon disappears, the perforation in the drum closes, and the hearing is completely restored.—*Therap. Woch.*

Hiccough cured by Traction of the Tongue.—Professor Lépine, of Lyons, states that he has arrested and cured a case of hiccough of four days' standing by drawing the tongue out and keeping it outside of the mouth for a few minutes. He does not know whether the procedure is of any practical utility in such cases, but theoretically it is of interest. It may be asked, if rhythmical tractions of the tongue excite the respiratory centre, as they do in restoring respiration in cases of asphyxia, how can they inhibit this centre, as in the case observed ? This apparent contradiction is explained by some experiments made by Professor Lépine, showing that the result of excitation depends, in the greatest part, on the state of the nervous apparatus concerned ; so that, while excitation of the nerves at the base of the tongue will excite the respiratory centre when this is paralysed, similar excitation will depress it if it is in a state of exaggerated excitability.—*La Sémaine médicale.*

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NOTES FROM THE CLINIC

OF

JONATHAN HUTCHINSON, LL.D., F.R.S.

Xanthelasma Palpebrarum.

By the term xanthelasma palpebrarum we recognise a common group of cases in which yellow patches, looking exactly like chamois leather let into the eyelid, occur. The woman shown presents a very good example of the disease, and conforms in all particulars to what we know respecting this symptom. The occurrence of xanthelasma patches on the eyelids must be taken separately from general xanthelasma or xanthoma multiplex, as it is sometimes called, affecting many parts of the body. Xanthelasma palpebrarum is very common, and I do not doubt that it is allied to the other form ; but xanthoma multiplex indicates organic disease of the liver, whereas the kind you see in this patient shows that there is only functional disturbance of the liver, that is, repeated functional disturbance, which has usually been denoted by a sick headache. These sick headaches, while revealing what is past, predict nothing as to the future. Very often those who have suffered from sick headaches in early life get rid of the liability as they advance in years, when the reflex susceptibility of the nervous system, and the ease with which nutrition is disturbed in the nervous system, gets less and less. I take it that adolescence is the period of greatest susceptibility to these sick headaches. The occurrence of sick headaches, therefore, does not mean that there will be progressive liver trouble. In xantheloma multiplex there is permanent disease of the liver. In nine cases out of ten the xanthelasma patch begins on the left side, on the upper eyelid ; then probably one forms below the canthus, then on the right side. The patches on the left are usually larger than on the right, and the yellow spots may extend until they curve round the whole of the inner canthus. The woman before you, for

whom we are indebted to Mr. Hopkins, has suffered very much from headaches in early life, but does not have them so frequently now. You will notice her sallow complexion, and she has, just above the inner canthus of the eye, on the upper lid, a characteristic yellow patch ; the area involved on the right side is slightly greater than on the left. The condition is not transitory ; I do not know that it ever disappears, but the patches do not increase after the symptoms reach their acme. If patients wish to get rid of the patches, they can be removed with the greatest facility, by snipping out the yellow patches and putting in a thin suture. Unless the patient is very young when the operation is done, the patches will not recur. I operated on a near relative of mine for the same thing, but the area affected was about five or six times as large as in this patient. She had suffered dreadfully from sick headache. I snipped out a number of them, and she has remained twenty years without the slightest trace of any return, and has lost her headache. My last case was an American professor, of very studious habits, who had very much deeper yellow patches, but did not know they were there, and assured me that they must have come very recently. His wife was not aware of their presence either ; but a daughter, who was much more observant, said her father had had them as long as she could remember.

I have the opportunity of demonstrating another very curious fact from a different patient, namely, increased pigmentation around the canthus. It is a disturbance of nutrition of the skin, and the origin is more serious than the pathological change which takes place in the skin. A fatty matter stained with bile is secreted into the structure of the skin, and it is curious how liable this area is to disturbances of pigmentation. In these cases, added to the history of sick headaches, is the statement that the attacks were accompanied by a peculiar discolouration. Some people habitually become dark around the eyes when they feel unwell, while others show the same darkening irrespective of headache. In this matter, of course,

the natural complexion of the individual must be taken into account. The disturbances of health may cause enlargement of the sebaceous glands, causing a mixture between a comedo and xanthelasma patch. Here there is not a flat yellow patch, but a number of yellow spots with black centres in some, implying that they contain sebaceous plugs. There is yet another group of cases, in which not the sebaceous glands but the sudoriparous glands are involved, giving rise to hydroa cystoma, with distended, chronic, persistent bullæ developed in the xanthelasma position, quite symmetrically, with a little ordinary chamois leather xanthelasma. The chief part of the disease is a watery cyst, developed, no doubt, in connection with the sudoriparous glands, with obstruction of its orifice and accumulation of sweat behind it. This patient here had suffered dreadfully from attacks of liver disease, and her sick headaches were of fearful severity, sometimes even producing temporary blindness. In the case in the bed here, I have squeezed one of the nodules, and proved that it contained sebum. Persons having comedones are very much less subject to liver disease than those in whom they are not present. I do not wish to state the case as regards sick headaches too positively. My American friend, to whom I have referred, told me he had never had a headache in his life; but he was of sallow complexion, and I ascertained that his general health had been disturbed, probably on account of sluggish liver.

I would also remark that the tendency to these patches is sometimes hereditary, and may not then be preceded or accompanied by liver disturbance. My observations, however, apply to the general run of cases.

Gout.

This man has had many attacks of gout, and his father suffered from true gout. Our patient has been a coachman, and has been very temperate in his early life, having consumed only a small quantity of beer. His attacks of gout commenced at the age of 22. When gout occurs at an early age it is always inherited; perhaps when gout occurs at all it is always inherited. I should doubt whether any individual could invoke gout by any diet whatever unless it was inherited. True family gout is indicative of inheritance. Without

this predisposition people may live just the kind of life which is supposed to produce it and not get the disease. "It takes three generations to breed a gentleman," is a trite aphorism, and I may add the words "and gout." The formation of urate of soda in the joints is a very special thing indeed, and comes about only by heredity, often quite irrespective of the habits of the person on whom the mantle has descended, but slight errors in diet may manifest the disease, even though the patient may have been most careful in every other respect. For instance, many total abstainers suffer from gout. This man knows his father had gout. A man once told me that gout had been in his family 200 years. A curious point about the case is that although his hands have become crippled and his fingers are stiff, he could not chalk with his knuckles, because there is no urate of soda there; he has not even the *nodi digitorum* nor enlargement of the phalanges. This is a typical case of true gout, and yet the arthritic inflammation has not produced chalk-stones nor any outgrowth of bone, but has caused ankylosis and destroyed the cartilage. There is no evidence of a tendency to the formation of tophi in the joints. So that you may have gout without the deposit of urate of soda. The latter condition is a very important one, but if we said it was an invariable feature of the ailment we should be laying too much stress upon it. With gout there goes a tendency to arthritis, to inflammation of a great number of joints, attacks recurring over and over again, excited and influenced by diet and by the ordinary conditions which induce rheumatism, namely, exposure to damp and cold and sudden changes of weather. But gout differs from rheumatism in this: the rheumatic tendency is fundamental, it is present in gout and in all of us more or less. Gout must be divided into special forms, some being attended with free deposit of urate of soda in the joints, and others not so. You will notice that this patient's toes and ankles are ankylosed very firmly, while his knees can only be slightly bent. I believe gout and rheumatism go together; you never have gout without rheumatism, though you may have rheumatism without gout, and gouty subjects suffer from arthritis from rheumatic causes. The patient now tells us that he had rheumatic fever when he was 25 years of age, at

which date all his joints were affected, and he was twelve months without being able to get about at all at one period of his life. Such a long spell is very unusual in cases of true gout; a great number of subjects of true gout are laid up ten days or a fortnight at a time, and get about between the attacks. There is thus a large element of rheumatism in his case.

So far we have simply accepted his statement that he has gout, though some patients distinguish very carefully between gout and rheumatism. An isolated attack in a single joint, generally the great toe, is usually gout; rheumatism very seldom attacks one joint only. The pain caused by gout is very severe, and then passes off. Apart from that, you will see he has a deposit of urate of soda on his tibia, and one or two on his ear. The patient is 56 years of age, and he has a very large arcus; his complexion is florid, and he looks in good health. You could not have a better instance of the occurrence of rheumatism and gout in the same patient. In all cases of gout we carefully examine the pulse and the urine, to see if there is any change in the arteries or kidneys. In reply to our question, he says he has been fond of strawberries and oranges. There are some curious problems as regards free eating, and I would give this hint. If a man will be a teetotaler he may eat fruit; if he drinks wine he must not have fruit. He must not mix fruit with beer or wine. Moreover nothing is more likely to produce gout than sugar and fruit. Conversely, a man may take a moderate amount of alcohol if he will abstain from fruit to which sugar has been added,—of course I mean grape-sugar; a certain quantity of cane-sugar may be taken with impunity. The worst fruit which a person with a gouty tendency can take is gooseberries, even though he may be an abstainer from alcoholic drinks. The few cases of total abstainers being liable to gout which I have known have all been large fruit eaters. One man in particular used to suffer from very severe attacks of gout, though he had been an abstainer all his life, and he was a most immoderate fruit eater.

Epithelial Carcinoma of the Tongue.

This man has an epithelial carcinoma of the tongue, far back, with enlargement of the glands under the edge of the sterno-cleido mastoid. The

case is interesting as showing what glands one must examine in carcinoma of the tongue. If the glands just behind the angle of the jaw be enlarged, the case is generally too far advanced for operation, because it is then most likely that the other glands in the neighbourhood which are not so easily accessible are already involved also, and that the disease would return. Then one should examine under the jaw, and the best way is to put one forefinger into the patient's mouth under the tongue, and another under the body of the jaw, to ascertain whether the submaxillary glands are affected. In this patient the glands are enlarged in both those situations, so that operative interference cannot be entertained. In a case of carcinoma of the tongue where the glands are not enlarged the growth should be removed promptly, and there is very good hope of the disease remaining absent. I have under care now a gentleman, a part of whose tongue I removed at least ten years ago. The case is interesting because he has a return in his glands, and that under the sterno-cleido mastoid was as large as a hen's egg; I concluded it was crammed full of epithelial cells. As only this gland appeared to be infected I took it out, and he has remained free from a recurrence for eight years. His case, however, is exceptional. On another occasion a man came to me with such a growth on the tongue, and the glands were implicated to a limited extent. I removed a large part of the tongue and the glands at the same time. I warned him of the probability of a recurrence, and within three months it occurred, and I repeated the operation. After that the other glands became implicated, and I did a third operation on him; that was six months ago, and he is now quite well.

As to the period at which the glands become enlarged after carcinoma, one very interesting fact is that I once removed a small portion of a man's tongue for an epithelial growth not larger than a sixpence under his tongue. He had been advised to have a tooth out, and was told that the place would heal up; I told him he had better have it cut out. It proved to be epitheliomatous, and quite two years afterwards he was in perfect health, without any recurrence, and we thought we were safe. But after that his glands enlarged, those behind the sterno-cleido mastoid and some which were deeply placed, so that I did not venture to

attack them. The cases afford an instance of the possible latency of infective matter; the recurrence was on the same side as that from which I removed the glands, and I have no doubt the glands were diseased from some material they had taken from the sore caused by the removal two years previously, but during that time they had shown no signs of growth.

A Case of Acne.

This patient comes to show us acne on his face, with a tendency to acne tuberosum, *i.e.* hypertrophy of the end of the nose, pustular acne, and rosaceous acne. We recognise these two forms of disease which acknowledge different causes, but which are yet allied, and very often run into each other. Of course, the most common form is acne vulgaris, which is incident to those who have a coarse skin and large sebaceous follicles, and it occurs in connection with reflex disturbances of health, and in nineteen cases out of twenty it is allied to affections of the sexual system. It usually decreases in degree as the reflex susceptibility diminishes. People who have very thin skin do not have acne; the disease is only possible in those who have large sebaceous follicles. The acne of adolescence, or common acne, occurs in many varieties; in some cases it is pustular, in some it forms abscesses, and in others lichenoid papules. As life goes on, there is a tendency to tuberous enlargement of the end of the nose, which may develop to an extreme degree, when it gains the name rhinophyma, and is accompanied by dilatation of the blood-vessels. This patient shows a little tendency to all those conditions; he has evidently had common acne in his early life. Different methods of treatment are indicated in different cases. One must attend to the patient's health, give aperients, regulate the diet, and use caustics. Sometimes cases in an early stage are more instructive than extreme cases. The disease is very hereditary; acne rosacea generally occurs in dyspeptics, is often due to errors in diet, and a frequent cause is intemperance. The occurrence of acne, however, does not by any means imply intemperance in every case. What we may term the red-nosed acne is very much influenced by exposure to cold, together with the consumption of stimulants. If a patient takes alcohol it diffuses the circulation of the

blood, preventing the contraction of the arteries. If a man were to go out on a cold night while half intoxicated, his face would not be rendered pale by exposure to cold; the capillaries would remain dilated. Possibly he takes a dram to "keep out the cold," but his capillaries become paralysed. Those who are disposed to this redness of the nose, and whose occupations cause them to be out in the cold a good deal, are in the habit of taking spirits while in the open air, and are very likely to get the extreme forms of acne.

ON CANCER OF THE RECTUM.

BY

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THIS patient is a farmer, aged 35, who has been sent to us because it is thought he is suffering from disease of the rectum. Before proceeding to examine him, we will ask him certain questions, his answers to which will throw much light on the case.

We must ask about the size, the shape, and the consistence of his motions, and also whether they are broken up. We must also ask whether the alvine evacuations contain anything else than faecal matter—mucus, blood, altered blood, or pus; whether they are passed with difficulty, and whether the patient has experienced any loss of power of control over the rectum; and then we must ask him whether he has pain either at the time of or after defecation; these points have all to be considered. First, then, we want to know whether the motion is smaller than normal, from which we might infer that there is some condition which prevents him passing a large motion. He tells us that about a fortnight ago he passed a motion of full size; he adds that he has often noticed the motion was flattened or grooved on one side, and that it is mixed with a good deal of slime. There is no history of loose motion, in fact on the whole his motions have been characterised by not being loose. He also says that the motion does not always come in one piece, but on being pressed, and asked if it is broken up "like a sheep's motion,"

he says "Oh, no." He first noticed blood with the motion last November—eight months ago,—and he says that since then he may have had a day pass without blood being noticed, but this has been rare, usually the amount has been only a little streak or tinge. The patient has told us that he passes slime with his motion, and now he adds that he passes a little slime several times a day, and this, too, apart from the acts of defæcation. As regards incontinence of motion and pain, he tells us that he can always hold his motion, but a little time back he was not able to hold wind, and that he has pain, which he describes as of a scratching kind, which lasts for about two hours after passing a motion; he feels it mostly in the bowels, but never anywhere else. Sometimes, he says, he passes what should be one motion in two efforts; as a rule his bowels act, he says, twice a day. These symptoms have lasted from three to four months; these, then, are the main rectal symptoms. What are the other symptoms we make out before we go into the history? He has been losing flesh, apparently to the extent of 9 or 10 pounds during the last six months, and the inference is that he may have something in his bowel interfering with his general health, but on inquiry we find that he has been doing his full work. You notice that he looks in good health, his lips are red, and his colour is good, his muscles firm (but you must remember that he is a countryman), his appetite is good, and he says he enjoys his food. To learn how long these symptoms have been coming on, we ask our patient when he was last quite well, and he tells us that last harvest he was quite well, and the first thing he noticed wrong was the presence of blood in his motion last November. Previous to this his bowels have always acted regularly.

To sum up, then, he has daily hæmorrhage from the bowels, passage of mucus and slime two or three times a day, apart from defæcation, and a certain amount of pain which he describes as a "scratching" pain in the bowel, which lasts for an hour or two after a motion. I also draw your attention to the fact, without laying stress upon it, that the motion has been grooved or flattened. We have also to consider the fact that he has lost weight, and is not able to control his wind, though he has been able to control his motions.

We now proceed to examine our patient care-

fully, and first of all we examine the abdomen, because we want to know if he has any retention of faeces; the parts to be examined, therefore, are the sigmoid flexure, and on the other side the cæcum. On examining the sigmoid flexure it feels about the size of one's thumb, and the cæcum is not distended; therefore we conclude that there is no retention of faeces above the rectum. We also feel for enlarged glands along the external iliac artery, and at the sacral promontory; we notice the situation of the edge of the liver and its condition; we notice the edge is just below the ribs, and we find that the liver is not enlarged, downwards at any rate. The glands are not enlarged on either side. There is, then, no enlargement of the liver, no retention of faeces, and no evident enlargement of the lymphatic glands. The rectum must now be examined, so we place the man on his left side with his left arm behind him, the right knee is drawn well up, and we place the hips on one side of the bed and the head on the other side. We first examine the anus for piles, for the "sentinel pile" of a fissure, and for a fistula. In this case there is no sign of either of these conditions. The next proceeding is to pass the finger into the bowel, and to notice while so doing if there is any undue laxity of the anus—patulous anus as it is called—or unusual resistance to the finger from spasm of the sphincter, or excessive pain. Then passing the finger on and examining the rectum, we observe particularly whether there is any narrowing of the bowel, any projection into its lumen, any ulceration of its surface, any infiltration or hardening of the bowel wall, and whether the examination gives unusual pain, and when we withdraw the finger we shall also notice whether there is any blood or mucus on it.

In this case, half an inch within the anus, is felt a hardened portion of the bowel, not involving the anterior part of the surface of the bowel, and not extending up quite so far as the finger can reach. Its surface is ulcerated and warty, and the examining finger is streaked with blood; the examination does not cause much pain, and the rectum is not fixed posteriorly. What is to be inferred from these facts? This thickening of the bowel that we feel, is it due to inflammation or to a new growth? A very little experience enables anyone to pronounce, without

any hesitation, this case to be one of malignant disease of the rectum. But I want you to follow me in tracing out the grounds of this diagnosis, and not merely to accept my opinion. Inflammatory infiltrations of the rectum are met with mainly, if not exclusively, in three forms. In the first place there is submucous suppuration, which may be met with as an ovoid abscess projecting into the bowel, covered by oedematous mucous membrane, or later, as a submucous fistula with a more or less well marked linear infiltration of the submucous tissue. In this form of disease the mucous membrane is healthy, or at the most oedematous, not widely ulcerated and warty as in our patient, and we therefore exclude submucous inflammation of the bowel. The second form is the chronic superficial infiltration and ulceration met with in tertiary syphilis. In this disease irregular sinuous ulcers of the mucous membrane are found which on healing leave puckered contractions of the bowel—a condition at once distinguished from the single warty ulcer on the mucous surface without any evidence of cicatrisation or healing. The third and rarest form is also one that is occasionally met with in tertiary syphilis, in which the whole thickness of the rectum becomes the seat of a diffused gummatous infiltration. The whole circumference of the bowel becomes involved, and its lumen narrowed : the examining finger passes into an unyielding tube which feels like thick india rubber ; the mucous membrane is hardened and may be ulcerated. This, again, is a condition wholly different from that we have found in this patient. I have spoken of this as an infiltration of the rectal wall, and the hard base and edge of the ulcer is the certain evidence of this : this serves at once to eliminate all benign, non-infiltrating forms of new growth, and by the method of exclusion we arrive at the diagnosis of malignant disease of the rectum. But we can look at it from the positive side as well as the negative. The abrupt outline of this single infiltration, its ulceration, and the fact that the ulcer nowhere extends quite up to the edge of the infiltration, the warty surface of the ulcer, the hardness of the growth, and its extent,—forming an ulcer larger than a crown piece after a duration of about eight months,—all these point to malignant disease. The disease has evidently begun in the mucous membrane, and is a columnar epithelioma.

Having arrived at the diagnosis of malignant

disease, the first question we ask ourselves is whether the case is one that can properly be submitted to excision; but before we answer that question as to this particular case, it may be well just to state the grounds on which excision can be justified as a wise treatment in any cases of cancer of the rectum. In the first place the operation actually cures a certain number of those who submit to it ; that is to say, they live for years without any recurrence of the disease. In cases where it does not accomplish actual cure, it prolongs life ; the patient may not be cured, but he has a year, or two or three years, without any recurrence, and the patient gains that time ; and when recurrence takes place, the disease is less painful than it would otherwise have been. In some cases, then, you get an actual cure ; in a large number of cases you procure prolongation of life and diminution of pain, and these are the two facts on which the justification rests. That is one side of the account, and we must ask ourselves whether the operation is attended with such a risk to life as to contra-indicate it, and also whether it has collateral disadvantages that outweigh it. Excision of the rectum for cancer is a serious operation ; its mortality in the best hands is about 15 per cent. But what is the condition of the patient after the operation? Is he better off or worse than if no operation had been performed? The answer we must give to this question is that previous to recurrence of the disease, the patient is free from pain, from haemorrhage, and from the too frequent passage from the bowel. His rest is not interfered with, nor is his digestion injured by the use of narcotics. He has good power of control over motions of normal consistence, but as a rule, only imperfect control over wind and over loose motions. Should recurrence take place, there is said to be less pain attending it than if the disease is allowed to go on unchecked ; this is due to the division of nerves affected by the operation. Therefore we may put it briefly that in properly selected cases of cancer of the rectum, excision will cure a certain proportion, that in another large class life will be prolonged, the patient will be relieved from the acute suffering of cancer of the rectum, and when at length the recurrence does take place, it does not cause such suffering as when the disease is left alone. Unless special precautions are taken, stricture of the new anus occurs, but this is an

accident that the surgeon should prevent. The mortality on this operation is not so heavy as to contra-indicate it; the causes of death are: 1st, haemorrhage; 2nd, shock; 3rd, peritonitis; and 4th, retro-peritoneal suppuration.

Passing from the general to the particular, we must now consider whether this case is one that is suitable for the operation, and that depends upon the answers we can give to three questions:—(1) Is there a good prospect of being able to remove all the infected tissue? (2) Will the operation be attended with special difficulty or danger? (3) Are there any features of the case rendering the ultimate functional result more or less favourable? The probability of removing all the infected tissue is the great justification we always seek in operations for the direct treatment of malignant disease. In the intestine this resolves itself into a question of the length and the depth of bowel affected, and the existence or otherwise of secondary deposits. We must draw a very clear distinction between the importance of the length and of the depth of the disease of the bowel. It used to be thought that only the lower three or four inches of the rectum could be safely excised, but later experience has taught us that a much greater length of intestine can be excised, and that the success of these more extensive operations depends upon certain technical details. If, however, the malignant growth has extended beyond the wall of the bowel itself, the dissemination of the infective matter is so rapid and extensive that operation is certain to be followed by speedy recurrence, and is unjustifiable. The most important fact in our case is, therefore, that the rectum where diseased is still moveable over the sacrum, and there is no evidence that the growth has spread through the bowel wall. This fact, and the absence of any sign of secondary deposit, are the great points that lead me to recommend operation to this man. We have also no difficulty from the part, or the length, of the bowel diseased. There is every reason to believe that we can excise this growth widely without opening the peritoneum, and this is always an advantage, and the operation on this part of the bowel is simpler than on the higher part of the rectum. Moreover, the fact that the front of the bowel is free from disease is another favourable circumstance, and for these reasons: (1) By leaving this part of the rectum

behind we avoid the most difficult step in a total excision of the lower end of the rectum—its separation from the urethra and prostate (vagina in the female); (2) in excising the front of the bowel it is necessary to keep close to the bowel, and so recurrence is more liable to occur; and (3) it has been found that when a strip of rectum is left behind it forms a protection against stricture of the anus, and also leaves the patient with better control over his motions. We, therefore, in this case answer our questions thus:—(1) There is a good prospect of removal of all the infected tissue; (2) The operation will not be attended with any special difficulty or danger; and (3) the functional result will probably be specially good.

There are two cautions I should like to give you in arriving at a conclusion on this important matter. Remember that the rectum is so adherent anteriorly to the prostate or vagina that it is a very difficult matter to determine whether a growth in this part of the bowel has spread through to these adjacent structures. Remember also that detached nodules of disease are often found at some little distance from the main mass, especially above it, and you must therefore always be prepared to remove a greater length of bowel than seems at first to be affected.

The steps of the operation I propose for this man are these: I shall first of all split the bowel, longitudinally and posteriorly, by passing a sharp knife into the anus and well above the growth, making the point of the knife pierce to the sacrum, and thus cutting out to the anus—cutting through everything. Then with a pair of stout scissors I shall make a cut on each side from the anal margin forward to half an inch from the raphe, as I intend to leave the anterior side of the bowel intact. On each side the scissors will sink deep into the ischio-rectal fossa, and cut the inferior haemorrhoidal vessels. Seizing one side of the bowel with pile forceps, and drawing it down the ano-coccygeal ligament, the levator ani and the fascia above and below it are cut, and the subperitoneal tissue opened up. Guiding the scissors with the finger I shall then divide the bowel longitudinally well in front of the growth. The other side of the bowel will next be treated in the same way, and I shall then make sure with my finger that I have freed the bowel well beyond the disease, and I shall feel for any separate nodules

above the main mass, and if found extend the dissection well above them.

The part of the bowel to be removed being now detached posteriorly and laterally, is drawn down first on one side and then on the other, making good and firm traction, and is cut off above with a series of short snips, so that the branches of the superior haemorrhoidal artery may be cut one by one, and not several together, and as each artery spurts it is seized with pressure forceps, and when the operation is completed there will be several pressure forceps hanging out of the wound. These forceps will then be taken off one by one in the order in which they were applied, and if any vessel still bleeds, it will be secured by a ligature; as a rule, very few ligatures are required.

Hæmorrhage having been stopped, any wound of the peritoneum is looked for, and if found is sewn up, and it must be closed very carefully. The operation is completed by bringing down the bowel to the skin. At first, the mucous membrane used to be stitched to the skin; this left a pocket behind the bowel, which became filled with blood and serum, and generally became infected, and the result was that suppuration occurred which led to septic peritonitis, and the mortality was very great; after this surgeons gave up suturing the mucous membrane to the surface, and they simply applied disinfectants to the wound, which was allowed to granulate up. This is a slow process and very liable to result in stricture, so what we do now is to pass the sutures through the cut end of the rectum and all the deep surface of the wound through to the skin, in such a way that when they are tied the bowel is drawn down to the skin and there is no pocket left at all. In successful cases you get very rapid healing of the wound, and there is no infective inflammation behind the rectum or risk of retro-peritoneal inflammation and peritonitis. There is very much less cicatricial tissue in your scar, and there is much less chance of subsequent stricture.

The first limit of the operation was to those cases which were within the reach of the finger of the surgeon, and for a long time the operation stopped there. Of late years, however, much greater length of gut has been removed, and the fact that the disease is above the reach of the finger is not sufficient to justify a surgeon in refusing to excise the rectum. This high operation is done in different

ways, but the main feature of the operation is that the surgeon gains access to the part by removing the coccyx and even a portion of the sacrum; he may remove some six inches or more of the rectum, or only a shorter portion. In some cases the divided ends of the bowel are united by a Murphy's button or by some other plan of suturing, or the upper end of the gut is brought out at the upper part of the wound, and a false anus made.

It has been advised that previous to a high excision of the rectum an inguinal colotomy should be made. It is exposing the patient to the small risk and annoyance of a second operation, but in view of the great gravity of the whole procedure this is not of great moment if real good is thereby attained. By a previous colotomy the bowel below the colotomy can be rendered fairly clean, whereas if there is no colotomy done you are unable to clean the bowel. If a successful colotomy has been done, not only can the bowel be cleansed at the time but afterwards the faeces do not foul the wound. The artificial anus is used for defæcation, but the excision is done for freeing the patient from the disease altogether; the mere fact that you have to combine colotomy with the operation must not be urged against the value of the operation. In this particular case that we have before us now, we shall get the disease away by the old-fashioned excision.

You must in this case have been struck with the very healthy appearance of the patient; it is noteworthy that the man has had since November a malignant disease, that is to say for about eight months, and yet has gone on doing his work perfectly well, and looks now very healthy. I want to draw your special attention to this point for two reasons, in the first place because of the indication it affords that these malignant diseases are local and not constitutional. When I was a student this point was keenly contested, and the general idea was that malignant disease was constitutional and not local, and we were taught a great deal about cancerous cachexia and its value for diagnostic purposes. In a discussion at the Pathological Society about sixteen or twenty years ago, the battle was stoutly fought out, and a very strong case was made out for the local origin of cancerous diseases, and since then the opinion has been veering round more and more strongly until now it is the general opinion that cancer is local in

its origin. Secondly, as it is quite in accordance with experience to find a man in robust health to be the subject of malignant disease, we must never neglect to examine a patient because he looks healthy when we see him. This man is not old, and I can easily understand any one just hearing the symptoms thinking that he is suffering from "piles." Do remember that because a man looks well and is youthful, that is no reason why you should not expect malignant disease and examine the man carefully. This man was fortunately examined early and sent up here for treatment. It is also noteworthy how little pain this man has suffered. If I had asked you to write an account of cancer of the rectum you would no doubt have put in a good deal about pain and diarrhoea and the small stools, but this man is undoubtedly suffering from cancer of the rectum and yet there is not much pain, there is no straining, and he has none of the characteristic stools, he has no obstruction, and he has not had to give up his work. It is a very important thing to remember that there is a large group of cases in which the symptoms are slight and the suffering is almost *nil* until the disease is far advanced. It often happens that the only symptom is a little occasional haemorrhage; never fail to make a digital examination of the rectum in all adults who come to you suffering from a passage of blood from the bowel; if mucus is also discharged the probability of finding a cancerous growth is still greater. Do not think that cancer of the rectum is attended by marked symptoms in all cases so advanced as to make a diagnosis certain. An alteration in the shape of the faeces is put down as one of the most frequent symptoms in carcinoma. The most frequent alteration is to have the motions broken up; this indicates a circular growth, and a considerable narrowing. Very occasionally and rarely you meet with tape-like motions, but they are more often described than seen. Sometimes, as in this case, the patient tells you that the motion is indented, and no doubt a new growth may cause such an indentation.

A CLINICAL LECTURE ON A CASE OF CANCER OF THE LIVER.

Delivered in the Western Infirmary, Glasgow, by

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GENTLEMEN.—The case to which I wish to direct your attention to-day is that of the man, J. N—, aet. 48 years, who was admitted on November 22nd, and died on December 5th. You will remember that our clinical observations failed to determine a completely satisfactory explanation of the case, but now that the results of the *post-mortem* examination are before us we are provided with the solution of our difficulties. The man, as we saw him on admission, appeared fairly strong and well nourished. He admitted the existence of a certain amount of pain in his left side, and this, with some discomfort after food and slight loss of flesh, he had been conscious of for a period of six months. At no time had he vomited or regarded himself as seriously ill. Indeed, he had continued his occupation as an ironfitter until the day of admission, and it was only on the urgent representations of a medical man, who had examined him in connection with his complaint of dyspepsia, that he consented to come into the Infirmary. Physical examination, however, at once detected very manifest evidences of serious disease. There was, in the first place, a tumour in the left hypochondrium, which we regarded with great confidence—though, as the sequel will show, incorrectly—as an enlarged spleen. It occupied exactly the position of a splenic tumour, was influenced by the descent of the diaphragm, was freely movable on bimanual palpation, and was dull to percussion. No notch could be detected, but this, though a valuable sign when present, is by no means always to be recognised, even in considerable splenic enlargements. In the second place, there was extension downwards of the area of hepatic resistance and percussion, so that the lower border of the organ in the middle line was placed about $1\frac{1}{2}$ inches above the umbilicus; the projection downwards to the right of the middle line was rather less decided, but the liver could be

readily felt for some distance below the costal margin. There was also a distinct "lump" raising up the parietes, and about the size of a Tangerine orange, situated close to the hypochondriac border in the line of the right nipple. This "lump" moved with the excursion of the diaphragm; it could not be separated from the liver either by palpation or percussion, and we had little doubt that it was a projection from the upper or convex surface of that organ. The exact nature of the "lump" was much more uncertain. There was no sense of fluctuation, such as we should expect in a projecting hepatic abscess, nor did it give the fremitus which is sometimes obtained on percutting a hydatid cyst. It was, indeed, quite hard and painless, and both it and the liver generally could be handled without causing the patient to make any definite complaint. These facts seemed decidedly against the suggestion of malignant disease of the liver, in which the nodules are usually both painful and tender. The absence of any well-defined and irregular lower edge to the liver pointed in the same direction, and the existence of an enlarged spleen was recognised as a further fact against the diagnosis, it being a familiar clinical experience that splenic enlargement is but rarely associated with malignant disease of the liver. Jaundice and ascites were both absent, a condition, however, quite consistent with a diagnosis of malignant disease of the liver, though each is a frequent result of such disease. The condition of the patient's general health, too, had manifestly some bearing upon the suggestion that he might be suffering from malignant disease.

As I have already pointed out, the lump projecting from the surface of the liver suggested an abscess, a hydatid cyst, or malignant disease, and we shall consider these more in detail in the later part of the lecture.

A combination of enlargement of the liver with enlargement of the spleen might well be due to amyloid disease, which frequently affects both organs. The enlargement of amyloid disease, too, is, like that in our patient, a painless enlargement. But amyloid disease would not cause a localised lump on the surface of the liver; it affects all parts of the organ equally, producing a smooth and uniform enlargement. And when it is present it usually attacks not only the spleen and the liver, but also the kidneys and intestines, leading

in the one case to polyuria and albuminuria, and in the other to diarrhoea. None of these symptoms were present in the case we are now considering. The conclusion against amyloid disease was strengthened, too, by the absence of any fact in the history that would account for the disease. The patient had never suffered from any long-continued suppuration (such as may occur in phthisis pulmonalis, or in tubercular disease of bone), nor from syphilis, and these are the recognised causes of amyloid disease. Syphilis, too, may enlarge the liver by causing gummatous deposits in the organ, and by leading to increase of the capsular and interlobular connective tissue. There was no evidence of syphilis in any other part of the body, and the general bearing of the man inclined us to receive his statement that he had never contracted the disease. For the same reason we believed his word that he had never been in the habit of taking alcohol to excess. And this was a matter of some importance, for excessive spirit-drinking, as you know, may produce cirrhosis of the liver, and this, by obstructing the portal vein, may cause considerable splenic enlargement. No doubt in the majority of cases cirrhosis of the liver leads to diminution in the size of the organ; but this is not always so, and the liver may indeed be very definitely enlarged. Other aspects of the case, however, rendered it unnecessary for us further to consider the possibility of either syphilis or alcohol as a cause of the abdominal condition. There were, indeed, other evidences of disease besides those present in the abdomen, namely, in the right chest, and manifestly the conditions above and below the diaphragm had to be regarded as possibly related to one another. I remind you of the physical facts elicited in the examination of the right chest. Percussion of the right front detected dulness from the lower border of the third rib downwards, so that we had a continuous area of dulness from the third rib to the lower border of the hepatic dulness, this area measuring in the nipple line seven and in the middle line seven and a half inches. There was also deficiency in the movements of the right chest; it measured one inch in circumference more than the left; and over the dull area the R. M. and the vocal fremitus and resonance were diminished. These physical signs are of course those of fluid in the right pleura, but it was observed that whilst in front the evidences

of abundant fluid were so decided, clear percussion behind extended down to the ninth dorsal spine, that is, the dulness and deficient R. M. were limited to the extreme base. Evidently, therefore, if fluid was present it was not free to find its level, but was somehow or other restricted in its movement, and confined for the most part to the anterior portion of the pleural cavity. But it was necessary to regard the thoracic condition in the light of the disease which undoubtedly existed below the diaphragm. It was, of course, possible to suggest that an accumulation of fluid in the right pleura had depressed the liver, and so given to that organ a fictitious appearance of enlargement. The facts generally did not, however, support this view, and, moreover, such a suggestion would not explain the presence of the tumour which we regarded as an enlarged spleen, nor would it account for the "lump" which appeared to project from the surface of the liver. It seemed, therefore, much more probable that the primary disease originated below the diaphragm, and that the abnormal condition in the right chest was a secondary result. Hence we were thrown back upon the necessity of endeavouring to determine the nature of the disease producing the abnormal physical signs which we found existing in the upper abdomen. As we have already seen, some of these signs, and more especially the enlargement of the liver, together with the presence of a "lump" or nodule on its surface, carried with them the suggestion that the case was one of malignant disease of the liver. On the other hand, the facts above alluded to opposed this diagnosis, or at least did not support it; and as our notes of the case accumulated we found that the temperatures, though pursuing an irregular course, were often decidedly febrile, which is not usual in cases of malignant disease. A high temperature is, however, possible with malignant disease, and too much weight, therefore, must not be attached to the existence of pyrexia. Another suggestion, which to some extent arose from the presence of fever, was to the effect that we had to deal with pus in the upper abdomen, and that this was present either as an abscess in the liver or as a subdiaphragmatic abscess. In either case it could be conceived that such a collection of pus might not only explain the abdominal condition, but also account for the physical signs in the right

chest. Thus it might, by causing irritation in the right pleura, lead to the presence of fluid in the pleural cavity, or by accumulating below the diaphragm it might raise the vault of the diaphragm and so interfere with the action of the right lung, and cause the impaired percussion and deficient R. M. detected over the front of the right chest. And, on the whole, it seemed more likely that these effects would follow such a collection of pus as is now suggested, than that a cancerous mass would be so localised in the upper part of the liver as to raise the diaphragm and cause extensive dull percussion in the right chest. Yet there were difficulties in this diagnosis also. An abscess of the liver could scarcely produce the firm nodule we detected on the surface of the liver, nor was it easy to understand how it could lead to decided splenic enlargement. And though the patient many years ago had been resident for a time, first in Egypt and then in Buenos Ayres, he had never visited the tropical countries, such as India and China, where almost all the cases of the large or tropical abscess take origin. And he had now resided here for fully six years, enjoying good health until his present symptoms developed, so that hepatic abscess seemed an altogether unlikely event. The existence of a sub-diaphragmatic abscess might quite well have accounted for at least some of the abnormal conditions detected in the upper abdomen, and such a collection of matter, by pressing the diaphragm upwards, might also have explained the dulness in the right chest. The evidences of a sub-diaphragmatic abscess are often very obscure. They depend, to some extent, upon the exact situation of the pus, and upon the degree of thickening and the adhesions produced in the neighbourhood of the abscess; pleurisy at one or both bases is a not uncommon complication. Such an abscess is usually secondary to some other condition, such as a gastric ulcer, gall-stone, hydatid cyst, &c. No conclusive evidence of any of these conditions existed here. There had been no severe pain after food, and no haematemesis, as we frequently, but not invariably, find in gastric ulcer. Nor had there been the severe paroxysmal attacks of pain which indicate the passage of a gall-stone through the cystic and bile ducts, and jaundice had never been present. A hydatid cyst of the liver is an extremely rare occurrence

in this country, and there did not appear to be anything in the patient's history or symptoms to support the view that the case was one of this nature, except perhaps that the splenic enlargement favoured hydatid disease of the liver rather than cancer.

The clinical position, therefore, was this : that there was some definitely abnormal condition in the upper abdomen, and also in the physical examination of the right chest was certain, and it seemed probable that the thoracic condition was secondary to the abdominal. The nature of the disease in the upper abdomen was a matter of great difficulty. In spite of the high temperature, the apparent enlargement of the spleen, the absence of hepatic tenderness, and other facts already alluded to, the presence of a definite nodule on the surface of the liver made it impossible to exclude the theory of malignant disease of the liver. On the other hand, the possibility of a collection of pus situated below the diaphragm was recognised, though nothing like a confident conclusion on the point seemed possible. Under these circumstances, and recognising the fact that in the case of an abscess surgical measures might be of avail, it was decided to make an exploratory puncture. After a negative result in the right lateral region of the chest, the needle of the aspirator was introduced in the ninth intercostal space four inches from the spine. At first there was no result, the pleural cavity, at least in this part of it, appearing to be free from fluid. But on pushing home the needle so as to presumably penetrate the diaphragm, a considerable collection of fluid was tapped. This fluid was of about the consistence of cream and of a light chocolate colour. Its reaction was neutral ; sp. gr. 1050. Under the microscope it was crowded with large, irregularly-shaped cells, nucleated and highly granular. The quantity removed was 58 oz. After removal of the fluid it was found that the right lobe of the liver, with its projecting nodule, had passed upwards behind the lower ribs, so as to be entirely beyond the reach of palpation, whilst the physical signs in the front of the right chest, as well as the condition of the splenic region, remained without substantial change. It was concluded, therefore, that the fluid had been removed from between the upper surface of the liver and the diaphragm, and that

its removal had been followed by an ascent of the liver, the descent of the diaphragm presumably being prevented by adhesions or otherwise. The question of the nature of the fluid now arose. It was certainly not pus, nor was it the watery fluid to be expected on the puncture of a hydatid cyst. Was it possible for it to occur in connection with a cancerous tumour? No doubt it is common to find that cancerous nodules, as met with in the liver, tend to soften in their central portions, so that they may present, even when examined through the abdominal parietes, a sense of obscure fluctuation, and may exhibit on section the escape of a creamy juice containing cancer cells. But it was difficult to picture the state of matters which would allow so large a quantity as nearly three pints of fluid to be produced in connection with a cancerous tumour of the liver. Still, there were the facts. The puncture was made when our minds were hesitating between a diagnosis of cancer on the one hand, and abscess on the other. Now the fluid certainly was not pus, and the cells might well be cancer cells, though none of our pathological staff would venture a positive opinion. To gain every chance for the patient I requested one of my surgical colleagues to examine the case, but in the end no operation was attempted, and the patient died three days after paracentesis had been performed.

Fortunately we were able to secure a *post-mortem* examination, and from it we gain a satisfactory explanation of the case. Here, in the right lobe of the liver we find a most remarkable cystic development, which almost entirely replaces the right lobe and presses upwards the diaphragm. The diaphragm is adherent to the base of the right lung, and the pleural cavity is free from fluid. There are several of these cysts in the right lobe of the liver, the largest having a diameter of about 4½ inches. The inner wall of each cyst has an exceedingly irregular surface, and it is from these cysts that the fluid was withdrawn through the aspirating needle. There are a few cancer nodules scattered over the surface of the liver. The most prominent of these is situated on the convex surface near the anterior border. This is the nodule which attracted our attention during life. There are two features about this cancerous growth in the liver which

are unusual. The first is the collection of the cancer mass mainly towards the upper part of the organ, so that the liver was enlarged by it rather upwards in the direction of the chest than downwards in the direction of the abdomen. The second is this extreme cystic condition. It would appear as if a number of cancer nodules had become situated close together in the upper part of the right lobe of the liver, and that the process of central softening, common in cancerous nodules, had been carried to an extreme degree, so that large collections of fluid had been formed.

Thus it happened that a cancerous formation produced a collection of fluid which bulged

curvature of the stomach. The perforation does not lead into the peritoneal cavity, but into a tumour mass which passes upwards and to the left, and is adherent to the spleen, which is spread out over the upper surface of the tumour. This tumour manifests the cystic tendency exhibited in such an extreme form in the growth in the liver, and it, as well as the other formations, has the structure of a cylinder-celled epithelioma. It is noteworthy how this tumour mass in connection with the great curvature of the stomach and the spleen simulated an enlarged spleen, and so far established a presumption against the diagnosis of malignant disease of the liver.



Liver showing cysts cut open by a vertical incision passing from right to left.
From a drawing by Dr. Alex. Macphail.

upwards and compressed the base of the right lung, an event well within recognition as a possible result of abscess or hydatid cyst, but certainly quite unusual in cancer.

The cancer of the liver is here, as usual, a secondary growth, and the primary tumour, as is very frequently the case, exists in the stomach. It has the form of a nearly circular ulcer about two inches in diameter, with indurated edges, and in its floor an irregular perforation which passes through the entire thickness of the stomach wall. The ulcer is on the posterior wall of the stomach five inches from the pylorus, and its inferior margin just reaches the great

There is one other feature of the case which may be alluded to in a word. It is the contrast between the symptoms from which the man suffered and the physical evidences of disease detected on examination. Here is a man who had complained, it is true, for several months of loss of appetite and some pain after food, but who had not the slightest suspicion that he was seriously ill. Yet during this time a malignant ulcer had penetrated through the wall of the stomach, and the malignant formation had produced a considerable tumour in the left hypochondrium and extensive secondary developments in the liver. It is a very forcible illustration of the danger of falling into very

grave error if you neglect to make a thorough physical examination even in cases where the patient's complaint does not suggest any serious disease. Pain in the stomach and other evidences of dyspepsia may, as in this case, be comparatively slight; and vomiting and haematemesis entirely absent; even though a considerable cancerous growth is taking place in the wall of the stomach. The evidences of this disease may, indeed, be restricted to some slight dyspeptic disturbance with more or less decline of flesh and strength. Such a combination of symptoms coming on in middle life must always be subject to suspicion, and must claim careful and repeated physical examination. This is one of the practical clinical lessons to be learned from the case that has occupied our attention to-day. The facts of the case, as we observed them from day to day, you already have in your note-books. It will be well for you carefully to compare them with the results of the post-mortem examination. If you do this, and if you have followed the remarks I have made about the case, you will, I hope, remember it as a source of clinical instruction, which will remain with you as of permanent value.

THERAPEUTICAL NOTES, &c.

Sleeplessness.—It is unfortunate that the physiology of sleep is not better understood. It is even more unfortunate that what we do know about the phenomenon of sleep is not more diffused among the profession. How frequently, indeed, do we observe physicians who mistake unconsciousness for sleep, and who seemingly regard the goal of the application of the therapeutical measures as having been reached, when they have succeeded in rendering a tired, worn out, nervous individual oblivious to his surroundings by placing him in a condition which they call sleep, but which is simply unconsciousness. An experience of several years in insane hospital practice, presented to the writer, in a very strong light, the enormity of this error and its wide prevalence. It was no uncommon sight to see patients brought to the hospital in an unconscious state, due to the administration of narcotics; opium or morphine being the favourite

drug used. One case is worthy of mention here: a male, neurasthenic, harmless, but suffering from the exaggerated mental symptoms so marked in this disease, was accompanied by his physician to the hospital. The physician stated his reason for coming was that he considered it important to keep his patient asleep, so that he would not appreciate the humiliation of entering the hospital, and further, that inasmuch as he had not been sleeping well for some time, and that he was now sleeping, he considered the opportunity a good one, to let him recuperate. To this end the physician was at intervals giving hypodermic injections of morphine, and the poor, worn-out, narcotized patient, saturated with morphine, and believed to be asleep, was, as best he could, with his worn-out nervous mechanism, fighting for his life, which his respiration, weak pulse, and inhibited reflexes showed was at very low ebb. This case was not an exceptional one; it was, alas! too common an experience. The physician had lost sight of the very essential principle which should govern our therapy in the treatment of sleeplessness, and that is, that sleep has for its object the repair of the wear and tear of vital processes of life, and to insure sleep we must not interfere with these processes, which we do when drugs are given until sedation results.

There is another thing to bear in mind in the consideration of sleeplessness, and that is, that there is a source of irritation somewhere in the economy, which, if relieved, will be followed by sleep. Again, "an axiom" well worth remembering, is, that the more gentle the means employed to induce sleep, the more natural will be the sleep induced; and the more gentle the means employed, the more careful must we be to select the right time for their use. We believe that Dujardin-Beaumetz was right when speaking of the use of drugs in the treatment of sleeplessness. He said, "That for a drug to be hypnotic, it must imitate the natural condition of sleep, by effecting a lowered intra-cranial pressure, and that drugs which, though bringing about unconsciousness do not lower cerebral pressure, or which increase it, cannot claim to be hypnotics. Opium and morphine are objectionable on this ground. Drug treatment must put the patient in a position to go to sleep in a natural way, and not *put* him to sleep." Sedatives do this, but narcotics do not.

In the use of sedatives we must be cautious and not use them *ad libitum*. Each case is a "law unto itself," and as such requires patient and persistent study ere we commit the folly of giving a hypnotic, when more simple and efficacious methods would produce satisfactory results. You cannot cure sleeplessness by drug treatment; the drugs simply conserve nervous energy and act as valuable assistants to the building-up process, necessary to cure the sleeplessness. Sedatives act, as before stated, by placing the patient in a position to go to sleep, and nature does the rest. In our experience we have learned to rely upon the bromides, choral, cannabis indica, and hyoscyamus as sedatives, which, if judiciously used, bring order out of chaos. The bromides lower the sensibility of the brain, and thus promote sleep. The single salts can be used, but in the writer's experience, where a sedative is indicated in sleeplessness, it is better to combine them, and when there is any excitement add chloral. Cannabis indica is a sedative which is but little used by the general practitioner, and for the reason that it is misunderstood, misrepresented, and as a result never used as it should be. Clouston, Mathison, and Echeverria have taught us their value. Hyoscyamus is another sedative, the value of which is not appreciated, a drug which is endorsed by Budde, Brush, Kraft-Ebing as an hypnotic.—*Medical Herald*.

The Treatment of Alopecia.—Brocq, in a brief article upon this subject, recommends the employment of the following prescriptions:

B Resorcin	gr. iss
Hydrochlorate of quinine	...	gr. iij	
Pure vaseline	...	3j	

This is to be applied, covering only a limited area at any one application, to that part of the scalp which is devoid of hair or from which the hair is rapidly falling. If the falling of the hair persists after the employment of this prescription, it is well to incorporate with it 5 to 15 minimis of the tincture of cantharides, or in other cases the following may be used:

B Resorcin	gr. iij
Hydrochlorate of quinine	...	gr. v	
Precipitated sulphur	...	gr. xxx	
Pure vaseline	...	3j	

Should either of these ointments produce much irritation of the scalp, so that a burning sensation

is produced which is too uncomfortable to be borne, it is well to apply after their removal an ointment composed of 20 grains of borax to 100 of vaseline. After the irritation is relieved, more feeble preparations of resorcin and quinine can be employed. One prescription which may be used is composed of:

B Salicylic acid	gr. v
Resorcin	gr. iij
Hydrochlorate of quinine	...	gr. v	
Precipitated sulphur	...	gr. xxx	
Pure vaseline	...	3j	

And if this produces much irritation of the skin, the ointment of vaseline and boric acid already named may be employed, or one composed of vaseline with 1 or 2 per cent. of carbolic acid added to it.

Should the falling of the hair be associated with seborrhœic eczema, it is often well to employ a mercurial ointment. One of the best of these is an ointment of the yellow oxide of mercury varying in strength from 1 in 25 to 1 in 10, according to the severity of the trouble. This should only be rubbed upon isolated patches at a time. After it has been employed and an alterative effect upon the skin produced, a prescription composed as follows may be used:

B Resorcin	gr. iv
Salicylic acid	gr. vij
Pure vaseline	...	3j	

Should the condition of the scalp be excessively greasy, the ointment which has been previously employed and the natural oil of the skin should be removed by washing the scalp with a weak solution of ammonia or by using Castile soap and warm water. Under no circumstances should the oily preparations be used continuously without the scalp being cleansed every now and then by some such process.—*Journal des Practiciens*.

The Technique of Supra-pubic Puncture.—Dr. Von Dittel has tapped the bladder above the pubes considerably more than one hundred times. He washes it out by means of a two-way cannula, and then introduces a Jacques catheter (No. 8), the caoutchouc of which has the property of swelling up and so effectually preventing any escape of urine. The catheter must be changed at least once in eight days; its stopper is to be removed whenever the necessity for micturition is

felt, once at least every four or five hours. When introduced in this way the foreign body seems much less likely to induce vesical catarrh than if inserted *per vias naturales*; this is probably due to the absence of the bacteria of the urethra. The puncture has a great tendency to spontaneous closure, which is a manifest advantage when the indications for its employment have been obviated. Von Dittel has always operated in the mid-line, but of late Schopf has conceived the ingenious notion of a lateral puncture, whereby the rectus or pyramidalis is used as sphincter, and the permanent catheter done away with. One disadvantage of this method is that the puncture requires keeping open by the nightly passage of a sound or drain. Furthermore, Von Dittel has shown that the depth of the peritoneal pouches enclosed by the urachus, obliterated hypogastric arteries, and the epigastric arteries is very variable, so that in some cases but a very small portion of the anterior wall of the bladder is free from peritoneum. In such instances lateral puncture may lead to perforative peritonitis, and of this he records one fatal case. He has therefore abandoned Schopf's procedure, and reverted to his own former method. He has found, however, that the poorness in vessels of the linea alba sometimes leads to necrotic changes round the puncture, and therefore now adopts the plan, particularly in old people, of operating just at the edge of this tendon.—*Wiener klin. Woch.*

Rupture of the Quadriceps Extensor.—1. In recent cases in which there is not much effusion and the joint is apparently not opened, and in which the separated ends can be approximated and detained by suitably adjusted pads, the mechanical treatment may be carefully considered. In the hands of the intelligent general practitioner this method may be expected to bring about a complete recovery in the larger number of cases. From nine to twelve months will be required to re-establish fully the normal functions. 2. A too prolonged fixation in bed is unfavourable to an early recovery; therefore early massage and passive motion are strongly advised. 3. The skilled aseptic surgeon who primarily resorts to the operative method in suitable cases (but the age and vitality of each patient must be most carefully considered) may quite reasonably hope to obtain a better result

in a large number of cases, and save his patient three to six months' time. Catgut, kangaroo tendon, or silkworm gut should be used, and when there is much effusion drainage should also be employed. 4. When the separation is greater than one and one half inches, or when the case has not recovered under the mechanical treatment, the operation is indicated. 5. As the length of time required for treatment is a very important consideration, so the operative method, which has diminished this period and also succeeded in a larger number of cases without increasing the danger, will be more often indicated and more frequently applied in the hands of the skilled surgeon.—Dr. WALKER, *American Journal of the Medical Sciences.*

Acute Tonsillitis.—Professor Grasset gives the following formulæ:—Every two hours take a spoonful of—

Water	fl. 3 <i>iv</i>
Chlorate of Potassium	5 <i>j</i>

Gargle often during the day with a hot infusion, or with—

Naphtholate of Sodium	gr. v
Hydrochlorate of Cocaine	gr. <i>iiii</i>
Syrup of Currants	... ,	3 <i>j</i>
Water, sufficient to make 1 pint.		

Brush the throat two or three times a day with—

Glycerine	fl. 3 <i>j</i>
Borax	3 <i>j</i>

If the local condition appear serious, begin by touching the tonsils with a 10 per cent. solution of nitrate of silver, and spray frequently with carbolised water or a 1 per cent. solution of phenosalyl. If the pain is too great, especially in deglutition, apply three or four times a day—

Glycerine	fl. 3 <i>j</i>
Hydrochlorate of Cocaine	gr. <i>viii</i>

For those subject to tonsillitis with large tonsils, outside of acute attacks, M. Grasset prescribes spraying the throat, morning and evening, with two teaspoonfuls of the following:

Hydrochlorate of Cocaine	gr. <i>viii</i>
Carbolic Acid	gr. <i>viii</i>
Glycerine	3 <i>vii</i>
Water, sufficient to make 1 pint.		

Or thoroughly brushing the tonsils every day or two with tincture of iodine and glycerine in equal parts.—*Consultations médicales sur quelques maladies fréquentes* (Paris).

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A CLINICAL LECTURE ON CEREBRAL TUMOUR, AND A CASE OF ARSENICAL POISONING.

Delivered at the London Hospital by

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GENTLEMEN.—We will first consider the case of cerebral tumour. The patient is a bargeman, æt. 36; there is no history of nervous disease in the family. He gives a well-marked history of syphilis; fifteen months ago he had a chancre, four months after that he was an in-patient with very severe secondary syphilis, so that there is no doubt about that disease. Whilst he was an in-patient he had a secondary eruption, *plus* deafness and a little left-sided paresis—that is to say, of the arm only. He went out very much better, and remained fairly well until August, 1895, the initial chancre having appeared in February or March of that year. Six months later he came in with headache (which was frontal at first, then general), accompanied by vomiting and vertigo. Two weeks later he developed paresis of the left arm. In September he had double optic neuritis; loss of power in the left arm; no loss of sensation; pupils equal and active; gait unsteady, especially when turning round, on attempting which he staggered and almost fell to the ground; but he could stand with his feet together and his eyes shut, so that there was no loss of co-ordination. The knee-jerks were very much exaggerated on both sides, especially on the left, and ankle-clonus could be obtained on that side. There was no albuminuria, and the specific gravity of the urine was 1015. The deafness on the left side had increased. He improved very much under iodides and mercury, and was discharged in October. He

remained fairly well until the end of March of this year. At that date, while doing up his boots, he had a fit and fell down, but did not become quite unconscious, and lost a considerable amount of power. He was admitted on April 23rd last, when he showed loss of power, without loss of sensation, on the left side, some incontinence of urine and faeces, and was fairly conscious. All the reflexes at that time were exaggerated, but more so on the left than the right side. He had no albumen or sugar in his urine, his lungs were normal, and he had no morbus cordis. He began to improve on iodides and mercury, when he suddenly had a relapse and became quite comatose, in which condition he remained with paralysis of the left side. Of course, we could not tell whether he had loss of sensation, nor could we be quite sure as to the presence of any paralysis on the right side, which showed a little tone. On the same treatment, viz. inunction of mercury and iodides, he improved. You will notice his present condition. He can walk fairly well, can move his arm on the right side perfectly well, and the left arm is recovering, though slowly, and there is some rigidity of that limb.

Now, the first point about the history is, that he has had syphilis, and signs and symptoms are present which prove pretty conclusively that he has had a cerebral tumour, so that the obvious conclusion is that the cerebral lesion is a gumma. The signs in our patient are simply those of intracranial pressure, without any definite localising sign or symptom, except that of deafness and the fact that the left side is more affected than the right. He has now, of course, the results of descending changes on his left side—that is to say, exaggerated knee-jerk, exaggerated wrist and elbow reflexes, and the ankle-clonus, which is usually obtained on the left, though it is sometimes also present on the right. I need not point out to you that the brain lesion is on the opposite side to that on which the disease is manifested, the fibres having decussated. Then there is the double optic neuritis, which has now gone on to some atrophy, but this is a case in which the sight

is very little impaired by the optic neuritis. You must always be on your guard in this matter, because though a person may see fairly well, he may still have double optic neuritis. It is said that the first trouble in connection with the eye is loss of colour vision, but he does not show even that. Such patients do lose sight altogether sometimes. I have mentioned that the pupils are equal and active, and there is still no loss of co-ordination with his eyes shut; his walk is more that of a debilitated individual who has gone through a long illness. He has now no headache, but has lost his appetite, which is, perhaps, explained by the large doses of iodides and the free use of mercury. Beyond that, the trouble is practically cured; he is even losing the signs pointing to descending trouble in the lateral columns. There is no loss of sensation. The case is a very fair type of syphilis as it affects the base of the brain.

Let us now consider what tumours may occur in the brain.

First, tubercular tumours, which usually occur at the base of the brain or in the cerebellum. You will remember how close the base of the brain is, posteriorly, to the tentorium, and so would be prepared to find an affection of the base spread to the tentorium and so to the cerebellum.

The second form of brain tumour, perhaps the commonest of all, is syphilitic. It may affect either the membranes or the brain substance, and may involve either the dura mater or the pia mater, and of course it implicates the brain substance if it affects the dura mater. It generally shows itself in either the convex portion of either hemisphere, or the neighbourhood of the sella turcica at the base. Speaking generally, the base is much more often affected than the vertex; even when the dura mater is affected, the statement as to locality holds good. When situated in the pia mater, the tumour is generally larger and softer than when in the dura mater. When the brain substance is involved, it is generally in one hemisphere, or in one of the crura cerebri. Syphilitic tumours of the pia mater are more common than in any other part. The lesions sometimes spread from there, so as to affect many of the large nerves, and often it is the implication of some large intra-cranial nerve which enables us to localise the lesion. Sometimes the disease spreads to the petrous bone and causes disease of

it, or simply deafness by affecting the soft portion of the seventh nerve.

There is another syphilitic disease of the brain which is not of the nature of a tumour, although it possibly might, in some cases, cause a tumour by producing an aneurysm; I refer to syphilitic disease of arteries. This is very common, perhaps more common in young people than gummatous tumours are. The disease simply causes a change in the internal coat of the artery, ultimately producing thrombosis, or is liable to do so, and will therefore be another cause of hemiplegia, but without the symptoms of cerebral tumour.

Then we may consider together the other intracranial growths. Of those which are called benign, we have fibromata and psammomata, and may almost include gliomata, because one form of them is to a certain extent benign. Then there are myxomata, which are very like soft gliomata, and all varieties of the more serious sarcomata (melanotic or otherwise) and carcinomata. Of these, sarcomata, gliomata, and psammomata, as well, perhaps, as fibromata, are mostly initial or primary in the brain, while carcinomatous tumours of that organ are nearly always secondary. I should doubt if carcinomata of the brain are ever primary, though one or two are said to have been reported. Gliomata and fibromata are mostly to be found affecting the connective tissue of the brain; a glioma is an exaggeration of the connective tissue of the brain. There are two types, one very soft, like jelly, and called net-celled; and a harder form almost exactly like round-celled sarcoma, with which it ought, perhaps, to be classed.

Again, entozoa will produce all the symptoms of cerebral tumour forming another class. You have to consider only two—*Cysticercus cellulosa*, and hydatid. The former has been found in the brain on several occasions. It varies in size from that of a pea downwards, and they are multiple, sometimes ten only, sometimes a hundred. They are dotted about for the most part in the subarachnoid space, the choroid plexuses, and the connective tissue of the brain,—not so much in the brain substance. The hydatid is a cyst which is nearly always single; you may find two, but generally only one. They occur, for the most part, in one or other hemisphere; sometimes in the ventricle, sometimes attached to the dura

mater. They may reach the size of a hen's egg, and present no peculiarities in the brain compared with their characteristics in other organs, except that they rarely undergo degenerative changes or suppuration. Their presence does not seem to cause any sort of irritation, and they are more commonly discovered after death without having given rise to any uncomfortable symptoms.

You may also put down aneurysms. Cerebral aneurysms are generally very small, though there are museum specimens as large as a Tangerine orange or hen's egg. Such large aneurysms, of course, do give rise to symptoms of cerebral tumour. The position of these aneurysms is at the base of the brain, where the large arteries enter. The large arteries of the circle of Willis or internal carotid might produce such an aneurysm, when, of course, the patient would manifest signs of intra-cranial pressure.

Now as to the symptoms and signs these tumours give, as compared with those presented by the patient I have shown you. Vertigo he had, and it is a symptom which is very common, but not always present. Vertigo is very often associated with a reeling gait ; this is more common with cerebellar tumours, and may occur only when the patient is walking, or when he is sitting still also. It is necessary to remember that vertigo is divided into forms : that in which the objects appear to move in definite directions, viz. from left to right or right to left, or upwards and downwards, or as if rolling away from or towards the patient. But do not forget that vertigo is caused by many things other than cerebral tumour.

The three next important symptoms, which we may consider together, are headache, vomiting, and double optic neuritis ; and they may be called the three cardinal symptoms of cerebral tumour. Any one, or perhaps even two of those three symptoms may be absent in cases of cerebral tumour. Headache is more often vertical, though it may be in the position of the tumour—frontal, vertical, or occipital ; it may be at the back of the eyes, or it may be general, as if the head was splitting. Headache is very varied in character, but it is a very constant symptom indeed, and long-continued intense headache is always a very suspicious sign.

Vomiting, either persistent or periodic, is almost always present ; it is known as cerebral vomiting,

and differs from ordinary vomiting in that there is no feeling of nausea associated with it. The patient simply opens his mouth and the stomach is emptied like a garden hose, with very little retching.

Double optic neuritis is a usual symptom of intra-cranial tumour, but it is sometimes absent, and it is sometimes present when there is no cerebral tumour. The three cardinal symptoms I have mentioned, however, when occurring together are practically conclusive of the presence of an intra-cranial tumour. A slow and irregular pulse is sometimes very marked in such cases ; occasionally the rate will be 30, 35, or 40 per minute, but later on, when the patient gets bed-ridden and more or less stupid intellectually, the pulse may become very fast indeed.

Then we come to that group of symptoms which helps us to localise the trouble—that is to say, those which show that some part of the brain has lost or has exaggerated its function ; paralysis, or paresis, or anaesthesia is the loss of function ; and convulsion is an exaggeration of function. In addition to these, which of course vary according to the position of the tumour, there are affections of special nerves. As I have said, the position of the tumour is often at the base, and the cranial nerves emerge from the skull at the base, so that it is not surprising to find them involved. There may be loss of smell, there may be double optic neuritis, and affection of the third nerve influencing the various muscles which it controls, so that there may be nystagmus, diplopia, or squint, or inability to move the eye in certain directions. There may be an affection of the fifth, resulting in anaesthesia of the cornea ; in such cases care must be taken to keep the eye clean, or the patient will have corneitis. Again, one part of the seventh nerve may be affected and cause deafness, while interference with the other part of it will cause facial paralysis.

In addition to these special nerves being implicated, the intellectual or emotional character of the patient may be altered. Of course, cerebral tumour is a very coarse change, yet for the most part it does not result in insanity. We know very few morbid changes in the brain of which we can say, "this is to be found in insanity." Subjects of cerebral tumour, however, very often have outbursts of passion, and towards the end they always become more or less demented.

Then follows obstruction of the venous flow. These tumours, being mostly at the base of the brain, are very likely to press upon the cavernous sinus or the internal jugular vein itself, and this pressure will stop the flow of blood, and will sometimes give rise to dilatation of the veins on the forehead or around the orbit.

Then there are trophic changes—bedsores and certain skin troubles, just as in ordinary hemiplegia, with loss of control over the sphincters, resulting in, first, a dribbling of urine, and finally, complete paralysis of the bladder, with overflow and distension.

I do not think anybody would mistake cerebral tumour if all the symptoms I have mentioned were well marked. But in some cases there are very few of those signs and symptoms present, and one has to be very careful to distinguish cerebral tumour from some other affections. The chief difficulty, however, is finding out where the lesion is. Localisation may be divided into vertex and base. Remember that a cortical lesion will nearly always produce some form of epileptiform seizure, some form of Jacksonian epilepsy. The epileptiform convulsions will possibly begin in the hand or face, travelling up in the way which is familiar to us. Therefore, with a cortical lesion it is important to ascertain what part was first and most affected, then one can localise by knowing the part of the brain controlling that movement. You ought, therefore, to remember where the various functions of the brain are located. I will indicate them to you on the board. You must also know the positions of the cranial nerves. If the lesion is in the medulla there will be loss of motion on the opposite side, and there will also be some symptoms of glossopharyngeal palsy; if in the pontine region there will be some form of what is called "crossed paralysis," that is to say, loss of motion on the opposite side of the body, and one or more of the cranial nerves on the same side as the lesion. For instance, if the lesion be in the lower part of the pons, there will be paralysis of the seventh; if in the upper part, paralysis of the fifth nerve on the same side as the lesion, and paralysis of the body on the opposite side. If it be higher up than that, for instance in the crus cerebri, the third nerve will be affected on the same side as the lesion, with paralysis on the opposite side of the body. All basal lesions are

localised by affections of the particular cranial nerves which are nipped in that region.

Regarding treatment in cases of syphilitic disease, it consists practically of iodide of potassium and mercury. You cannot do everything with iodide of potassium, but that joined with mercury will generally produce very excellent results. Sometimes it is a race between the medical man and the tumour. A man may be brought to you absolutely comatose, with lungs becoming oedematous, and in a state very near death. These syphilitic tumours grow very rapidly, but they also retrogress quickly under the classical treatment. But you must begin with large doses, giving more than in an ordinary case of syphilis, and rub the blue ointment into the axilla night and morning, getting in as much as you can. Perhaps the patient will remain in that comatose condition for a week; then you will see his intellect returning to activity, and after that improvement in the parts which were paralysed, beginning with the lower limbs. This man's leg recovered before his arm; you see his arm is still affected.

As to after-effects, these tumours tend constantly to recur. This man has now had three definite attacks of paralysis or paresis of the left side, with or without deafness, and that is the usual history. You can rarely completely cure them, because, probably, they cease to take medicine when they go away from the hospital. Continuation of treatment will probably keep such subjects fairly well, but when they are full of syphilis they are liable to syphilitic arterial disease. This man is also liable to other syphilitic troubles in his brain, because he has shown a tendency for his nervous tissues to be affected beyond his other tissues. Therefore, he may get the same arterial trouble, which may result in thrombosis or haemorrhage.

The interest of the other case I have to show you—one of arsenical poisoning—consists simply in its rarity. The man works in "sheep dip," a compound in which arsenic largely figures, and his history is briefly as follows:—He has not vomited, denies syphilis, and is not an alcoholic. Twelve months ago he was affected with what was called blood-poisoning, and was treated in the infirmary. He had an eczematous, almost an ulcerated condition of both arms, and was discharged practically cured. One month after being discharged he first noticed loss of power in the legs

and feet, and in a few days his hands and arms became similarly involved. Following the eczematous condition were all the symptoms of peripheral neuritis. He had been treated largely with lead lotion for his eczema, and it was thought by some, when he came in, that the case might be one of lead poisoning, not of arsenical poisoning. But the paralysis did not follow the usual position of lead poisoning, which is generally confined to certain areas, such as to the musculo-spiral. He had no headache, no vomiting, his pupils were equal and active, and his fundi normal. When examined he had numbness of arms and legs, feet and hands. He had paresis to such an extent that he could not walk, nor grasp a person's hand. You see he is now able to walk very fairly, and his grasp is moderately good. The knee-jerks were absent ; there was no ankle-clonus or other exaggeration of reflex noted anywhere. When we came to examine the numbness we found he had lost tactile sensation in the hands and feet, but he was able to appreciate heat and cold. As he lost no control over his sphincters, ordinary paraplegia was excluded. When we examined with the battery as to the nature of the muscular affection, we found that excitability to the electric current, whether interrupted or continuous, was very much diminished ; the reaction was extremely sluggish, but did not present the reaction of degeneration. Otherwise our patient showed every sign of peripheral neuritis. He has largely recovered on iodide of potassium. We had considerable difficulty at first in getting any improvement, but when he once began to get better he did so rapidly.

I would like to remind you that the chief causes of peripheral neuritis are : syphilis, alcohol, traumatism to the nerve, leprosy, *i. e.* the anaesthetic form, diphtheria and influenza, and certain mineral poisons, such as lead (perhaps the commonest), arsenic, or very large doses of mercury. You may not see another case of arsenical peripheral neuritis in your lives. Lead, mercury, and arsenic are the three chief poisons you have to think about in this connection. The symptoms are numbness and loss of power, and the peripheral neuritis is usually symmetrical. With the exception of lead poisoning, it is usually the legs rather than the arms which are affected ; whereas with lead the arm is more often implicated than the leg, and the paralysis is not so sym-

metrical, usually affecting the muscles supplied by the musculo-spiral, with the exception of the supinator longus. The knee-jerks and other reflexes are almost invariably lost ; the muscles waste and are very often tender to pressure, and for the most part they present some form of the reaction of degeneration to the battery.

In most cases the treatment is obvious. In syphilis, anti syphilitic treatment ; in alcohol, simply checking the habit will be quite sufficient. We have upstairs a man who was admitted as a long-standing case of tabes dorsalis, and who has been an in-patient of nearly every hospital in London. He is a chronic alcoholic, and has no tabes dorsalis whatever. When the knee-jerks are absent with a certain amount of added functional trouble, and the patient is artful, you may have the greatest difficulty in distinguishing the case from one of locomotor ataxy. In leprosy you cannot do much. All the mineral poisons will be helped out by iodide of potassium, and of course the patient must stop working in the particular poison. As to injuries, you have to get the injury repaired, and the patient will get well. A man falling to sleep with his arm over the back of a chair will get all the symptoms of peripheral neuritis, but the prognosis in such a case may be invariably favorable.

WITH MR. THOMAS BRYANT

IN THE

WARDS OF THE CENTRAL LONDON SICK ASYLUM.

LADIES AND GENTLEMEN,—You are quite aware that in the clinical lectures or demonstrations given in this sick asylum we have to use the material which happens to be placed before us, as the field for selection is somewhat limited and some good examples of disease have already been utilised at previous lectures. I do not, therefore, propose to-day to give a set lecture, but to take the two cases which are at my disposal as texts, and to ask one of my hearers to assume, for the time being, that he is a student and I a teacher,

and so to the class, as a whole, make this the channel of instruction.

[We present our readers with the main points thus discussed.]

Our first patient is a man who is both deaf and dumb, and incapable even of communicating by signs, so that we are unable to get any help from him. The history we have of his case is simply this: that he is 60 years of age and was admitted two years ago into this house. He had been looked after previously by his sister, who reports that he was always rather of a simple character, and had been earning his living by shoe-blacking. She sent him to the infirmary as he was offensive owing to his inability to retain his urine. I have ascertained from my friend, Mr. Hopkins, that there is nothing wrong with his bladder or urethra, no stricture and no prostatic trouble; the incontinence of urine being apparently due to that atony of the bladder which is met with in old and imbecile men.

On superficial examination of the patient, you will at once see that it is a case of scrotal tumour, the swelling in the scrotum being large, and extending up the left inguinal canal as far as the internal ring. At the first glance of the case, therefore, an inguinal hernia is suggested, but should you on digital examination find that the inguinal canal is not really involved, we shall know there is no hernia. If, on the other hand, you find on examination that the scrotal swelling does involve the inguinal canal, it does not follow that the trouble is a hernia, for it may be a hydrocele passing up the canal, for such a tumour may extend as far as this on account of the tunica vaginalis being open to the internal ring. Or it may be a haematocele from the effusion of blood into the cavity of the tunica vaginalis. Now let us examine the patient manually and see if this mode of examination gives any support to the view which mere inspection of the case suggested. On doing this, it was at once found that the swelling in the inguinal canal was not real, although apparent, and that the scrotal tumour was limited to the scrotum, and was not therefore a hernia. Now what may be the nature of the scrotal tumour? Roughly it may be said that it may be an enlargement of the testicle from inflammatory action, syphilitic or otherwise, or from the presence of some new growth, such as enchondroma, malignant tumour, or cystic disease;

or it may be a hydrocele or haematocele. I told you he had been here two years, and that he had probably had the tumour several years before that, as it has not appreciably increased during his stay. This fact—its long duration—consequently excludes malignant disease, although it does not exclude either enchondroma or cystic disease. Moreover, no inflammatory condition would probably remain stationary for two or more years. A suspicion that it is syphilitic in its nature is justifiable, perhaps, but in two years a syphilitic testicle would have probably broken down, or the organ would have been atrophied. We next have to ascertain whether the tumour is firm and solid, or whether it fluctuates, and we find that it is moderately firm in some parts, while in others it is softer and fluctuates. The outline of the tumour is likewise smooth.

With these facts before us a hydrocele of the tunica vaginalis is suggested, or a cystic hydrocele or spermatocele, or it may be a haematocele. To go further for diagnostic purposes, an exploratory puncture into the tumour should be made, when if the fluid prove to be of a pale amber colour, a vaginal hydrocele would be diagnosed; if of a water colour, a cystic hydrocele; and if of a slightly opalescent character, a spermatocele, for such a fluid contains spermatozoa, and looks like water into which a little milk has been dropped. A few years ago I had a man with three cysts connected with his testicle; two of these contained spermatozoa, the third was watery and was free from them. The man had previously had a blow upon the enlarged organ, and it was probable that at the time a tube had accidentally been burst into the cyst. So that a cystic hydrocele is not always connected with the spermatic duct, although anatomically it is usually connected with the epididymis. You must consequently bear in mind that if the disease be cystic you will find the testis in front; if of the tunica vaginalis it will be behind, unless in exceptional examples.

The next step to be taken for diagnostic purposes would be to examine by electric light to see if the tumour is translucent; should it be so, the diagnosis of hydrocele would be fairly complete; should it be otherwise, the tumour may still be a hydrocele, although with greatly thickened walls, or a haematocele. In the case before us there is no translucency; it is, therefore, either a hydrocele

or hæmatocèle. Now, a hæmatocèle will rarely remain so stationary as this has been ; the blood-clot which has been formed either goes on organising or it will break down and form some kind of inflammatory tumour,—and a hydrocele, as a rule, goes on increasing. I will now, therefore, anaesthetise the organ with ethyl chloride at one spot and puncture it with a needle ; to do more I am not permitted, for the man's sister positively forbids surgical interference. You see I am unable to draw off any fluid ; it has been almost a dry tap, and yet I feel convinced that the case is one of chronic hydrocele with very thick walls, in which the fluid is too thick to pass through the fine cannula.

The other patient I wish to show you is a man æt. 61, who was admitted ten weeks ago in his present condition. The history is, that eighteen days prior to admission, whilst intoxicated, he was run over by a four-wheeled carriage, the wheels of which passed over his neck. Before the accident his neck was straight, and natural in appearance. He was subsequently at a workhouse a fortnight, and after four days wandering about he came here. You see there is a deformity of the neck with limited movement, the bones projecting to the left, forming a convexity on that side, and tilting the head a little. As we look at the case it would appear to have been one of fracture and displacement of the cervical vertebrae, and yet it is difficult to understand how this accident could have taken place without some evidence of injury to the spinal cord or to the nerve trunks given off from it, and yet on inquiry it seems there was not at the time nor since, any symptoms to indicate that either the cord or its nerve trunks were involved. There was no paralysis. The first striking point on looking at the man is that he moves his head as a whole, as if all the upper bones are ankylosed ; where rotation takes place is between the sixth and seventh cervical vertebrae. He can, however, nod his head. It is probable that in this case the laminae of the vertebrae have been broken and displaced without injury to the cord. I have a case at present under observation which bears upon this.

A woman, 30 years of age, while walking along the street, saw a boy about to be run over. She ran and pulled the boy away, but she was knocked down and the wheels went over her right arm,

right clavicle, right side of her neck, and lower jaw. As a consequence, she had broken ribs, with injured lungs, a broken clavicle, and her jaw was fractured in two places. The cervical vertebrae were turned to the left, and she had paralysis of the muscles of the right shoulder and anaesthesia of the left and upper part of her chest. With these multiple injuries one naturally thought she would die, but she did not. The jaw was bound simply with a bandage, for to do more was impossible, as she had pneumonia as a result of the injury, and the head was kept as quiet as possible with sand-bags. In a month's time the paralysis of the arm disappeared altogether, and within three weeks sensation returned in the left arm and chest. As she required as much air in the lungs as possible, we left the jaw to take its own course. Later it was wired with a Hammond splint, winding the wire round three or four of the teeth. That helped us very much. There was a little necrosed bone subsequently, but that was removed. She is now well, with a crooked neck like this man, but without any real permanent injury to the cord.

Had any attempt been made after the accident to mould such an injured neck as this man's or woman's there would have been great danger of injuring the spinal cord, either directly by the broken bones, or indirectly by removing pressure from the large veins which surround the cord, and so giving rise to haemorrhage. The surgeon who had charge of the case probably did right in leaving the man alone, for in surgery action is not always beneficial ; to stand still is sometimes better.

When I was a student and was under Mr. Bransby Cooper's care, a case in point which happened is worth recording. It is that of a man who was brought into Guy's Hospital with a tremendous cut through his head by a circular saw, from the occiput over the vertex nearly to the nose, and one side of the skull appeared to be an inch lower than the other side. It was thought right to leave him alone, except to dress the wound and attend to his wants ; no hopes of his surviving the injury being entertained. He was watched carefully and with interest, until he got well, which he did in the course of time. The brain tissue, which escaped, apparently left no signs of weakness, for he had no paralysis. Any

attempt at readjustment of the parts in this case would have been probably fatal. If the woman I have referred to had been in very great pain I should probably have attempted readjustment of the displaced bones, unless pain could have been controlled by morphia ; but by trusting to nature more good is sometimes done than by trusting to arts.

A CLINICAL LECTURE ON PARALYSIS OF THE SOFT PALATE.

Delivered at the National Hospital for the Paralysed and Epileptic, Queen Square, London, by
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Physician to the Hospital, and to the Great Northern Central Hospital.

GENTLEMEN,—I propose to show you this afternoon some cases of paralysis of the soft palate. In the first case the paralysis is only on one side, and it is, of course, necessary to differentiate between this and the commoner form in which both sides are paralysed. This patient is 54 years of age, and follows the occupation of rag-dealer. Eighteen months ago he had an attack of right hemiplegia, which came on quite suddenly, but he did not lose his senses. He was in bed four weeks with loss of power in the right arm, right leg, and right side of the face, and his speech became affected and has remained so. I shall not now go into the cause of this, as I do not propose to deal with hemiplegia; but it is probable that his case is one of thrombosis, not haemorrhage. The second cardiac sound was accentuated; but no albumen in the urine. On examination the patient was found to have right hemiplegia, and paralysis of the right side of his face was extreme. He can now whistle, close the eyes, and show the teeth. A year ago he had some difficulty in putting the tongue into the right cheek, but he can do so fairly well now. I may here mention that in examining the tongue it is not sufficient to see whether it comes out straight; it should be ascertained whether the patient can move the tongue in

all directions, and especially whether he can put the tip of it into either cheek and elevate the tongue at the end. It is rather difficult to demonstrate the condition of a soft palate to a large number, but the palate is drawn up one side, the left, more than on the other, and the raphe is drawn over towards the left side. In well-marked cases there is a dimpling of the soft palate on the unparalysed side. You will notice that the paralysis of the soft palate is on the same side as the hemiplegia.

I regret that the next case * has not attended, and I must therefore be content with reading the notes about her. She illustrates in a double degree what this man has,—that is to say, she has paralysis of the soft palate on both sides, and probably from lesions similar to his. Her age is 41; she is married; no definite history of syphilis, but she had ulcerated throat eight years ago; has had one still-born child; two healthy children alive. She has had two attacks of paralysis; the first was in July, 1894. The night previous she went to bed quite well, but awoke in the night and felt her face drawn to the right side. She did not lose the power of speech, but her utterance became thick; there was no trouble in swallowing. She forgot names, but did not use wrong words. The second attack occurred in November of the same year, when she awoke in the night with a gurgling sound. She went to sleep again, and the next day she could not say a single word, nor even grunt. That is a very important fact. She was also unable to swallow properly, and had to be fed by a spoon. In a week she began to make some sort of noise, but swallowing continued defective, and sometimes food "went the wrong way." She could not open her mouth properly for a week, and was unable to move the side of the face. Two or three weeks later she began to speak, and she thinks the tongue went to the left on protrusion. In the second attack the right arm seemed heavy for a month; legs not affected; no anaesthesia. When I saw her a month ago she could purse up her mouth and evert the lips, but on the left better than on the right. She could retract and elevate both sides of the mouth, but not quite so well as normal; could close the eyes well, and the tongue

* This patient had another attack on this day, from which she died.

was protruded straight. There was no atrophy of the tongue; but she had a nasal voice, and talked like a case I shall show you presently. I found the palate did not move on either side on phonation, but it moved reflexly when tickled with a quill pen, when it was drawn up equally on both sides. She could not close the posterior nares, and was therefore unable to inflate the cheeks. On testing her reactions with the faradic current, it was very difficult to be certain, because the reflex was so well marked that the part was drawn up at once. The larynx was normal (Dr. Semon); tactile sensibility in the palate was also normal; no albumen in the urine.

I now show you a man æt. 66; there is no specific history. Nine months ago he began to have gradual numbness in the right side of his face and a drawing of the face to one side; he had also some difficulty in swallowing, and his speech became affected about three weeks after the onset of the attack. The onset in his case was gradual, thus differing from the other two cases—a difference which is very important from a diagnostic point of view. Six weeks ago he began to have enlarged glands on both sides of the neck. At present he has nasal speech, and is unable to close the posterior nares so as to inflate the cheeks. There is no movement of the soft palate whatever, either on phonation or to reflex irritation; the larynx is not affected (Dr. Semon). There is no reaction in the soft palate to the strongest faradic current he can stand; that is, a faradic current which causes contraction of the tongue and of the face will not cause contraction of the soft palate through the damp mucous membrane, whereas normally a weaker current would excite the palate than the other parts through the skin. To galvanism there is a slow reaction of the soft palate, the reaction to the anode being better than to the cathode; therefore there is the reaction of degeneration. In addition, he has some paralysis of the right fifth nerve—the motor part of the fifth and the sensory part of the fifth also. He has no sense of taste on the right side of the tongue, front or back. The olfactory sense is fair, but the right side is not so good as the left. There is no facial loss, and nothing amiss ophthalmoscopically. In testing the fifth nerve the best way to ascertain if the external pterygoids are acting equally, and whether the mouth is opened straight, is to see if the

interval between the two central incisors of the lower jaw part from their opponents in a vertical line in opening the mouth. When there is deviation due to weakness of one external pterygoid, the opposite condyle is pulled more forward, so that the lower jaw is carried over towards the paralysed side. Another test is that the muscles which close the jaw contract fairly well, but one masseter and one temporal is weaker than the other. The tactile sensibility is absent from the right face, but is present about half an inch from the edge of the anterior pillar of the fauces, so that the whole of the front part of the palate is anaesthetic,—that is to say, the roof of the mouth, the hard palate, and the anterior half of the soft palate is anaesthetic. The patient also has on the right side some conjunctivitis; the conjunctiva is anaesthetic, and the conjunctival reflex is abolished. The tongue protrudes straight, the larynx is not affected, nor are the sterno-mastoid and trapezius. You will notice that the patient can only inflate his cheeks when I compress the nose; that his speech is nasal, and that he has large fixed glands in his neck.

The last case I will show you is under Dr. Gowers, who has kindly allowed me to show the patient. He is undoubtedly a case of paralysis of the soft palate from a lesion of the bulb. His age is 46, and he came in on account of weakness and thickness of speech. A month before Christmas he noticed weakness of both hands, followed by weakness of the right arm. Subsequently he had to be careful when drinking, and avoided any cold liquids. Anything he drank appeared to regurgitate through the mouth and nose, while dry bread would stop in his throat. Just before Christmas he noticed that he was no longer able to whistle. He is unable to whistle because he cannot purse his mouth properly. His arms have wasted since Christmas.

Here is an intermediate case, where the soft palate moves slightly on either side, but not sufficient to close the posterior nares. He has reflex action of the soft palate to tickling, but slight; in the case of the other man it was completely abolished. The facial movements are preserved fairly well; the tongue is tremulous, but not atrophied. The hands are atrophied, particularly the first interosseus, as shown by the concave curve of the metacarpal bone, and in the ball of the hand.

He can neither abduct the thumb nor separate the fingers. Further than that, he has a symptom which at once puts the case into a definite category, *i. e.* increase in all his deep reflexes. He also has a symptom which I believe I was the first to point out some years ago, namely, lower jaw clonus. The first case of this I saw was in 1881, in a woman whose lower jaw was always going off spontaneously into rhythmical tremors. This patient has also clonus of the ankle, and it is what is known as amyotrophic lateral sclerosis, that is progressive muscular atrophy preceded by lateral sclerosis of the cord. There is atrophy as shown by the soft palate, due to actual disease of the cells, either in the anterior horns of the cord, affecting the hands, or in the bulbar nuclei, affecting the soft palate and the tongue; and along with that there is sclerosis of the lateral columns.

Paralysis of the soft palate may be due to several causes, and first it will be well here to review the distribution of the nervous tract, so as to trace the impulses from the cortex cerebri to the muscles themselves. Starting above, the large motor cells of the cortex send down fibres through the internal capsule, the crus, and the pons and medulla; these fibres then cross over to the opposite side, to the cells of the nucleus which supply the soft palate, and from the nucleus fresh fibres go to supply the muscles of the soft palate itself. The diagram I show you will explain this. Taking the cortex first, the point where you get, by electrical stimulation, movement of the soft palate on the opposite side only is at the foot of the ascending frontal convolution. Mr. Horsley and myself, working on the subject in the monkey, found that there were certain points at the foot of the ascending frontal convolution, stimulation of which gave elevation of the opposite half of the soft palate, *i. e.* stimulation of the left cortex caused drawing up of the palate on the right side only. I pass round a diagram which shows the result of our work. In each experiment a drawing of the cortex was made on paper ruled with lines which divided it up into squares of 2 mm. the side, and each of these squares was separately stimulated. The squares where the movements were obtained are marked in the diagram. The area for the soft palate is at the lower end of the ascending part of the frontal convolution, and close to the points which, when stimulated, pro-

duced movements of the face and tongue. I believe it had not been shown before that the movement is unilateral. The question arises whether a lesion will produce this paralysis of one half of the soft palate. It is probable that the soft palate fibres would go along with those of the tongue, the representation of which is close to that of the palate. The next question which arises is, where is this nucleus which sends fibres to the soft palate? It used to be thought that the soft palate was supplied by the facial nerves, and it was utilised as a means of ascertaining whether the lesion was inside or outside the skull. Dr. Hughlings Jackson said, years ago, that he had never seen a case of paralysis of the soft palate in paralysis of the facial nerve; and Mr. Horsley and myself did some experiments to test this. We removed one hemisphere and stimulated the peripheral ends of the cranial nerves, and found we got no movement of the soft palate except from the accessory nerve to the vagus; no movement from the facial or pneumogastric or any other nerves.

The next point is to diagnose where the lesion is. First, taking the causes of paralysis of the soft palate from below upwards, the simplest case is one of paralysis of the nerves, as after diphtheria. In this case there is the history of a sore throat or definite diphtheria, then gradual onset of paralysis, always bilateral. Reflex action and faradisation are both lost, and in many cases sensation is also lost. You will see why reflex action must be lost as well as faradisation, namely, because the lesion is below the nucleus, and the muscles are thus cut off from their trophic centres. If the motor or the sensory nerves be affected there is no reflex action, because the reflex arc is broken; and when the motor nerve is affected electrical reaction of degeneration ensues. In a lesion of the nucleus of the accessory nerve to the vagus the onset is, as a rule, gradual, as in the man I showed you with amyotrophic lateral sclerosis. In a case like that the onset is gradual, and is nearly always bilateral; that is, the nucleus on both sides is affected. There is another form met with in which the nucleus itself is affected, and which begins with a sudden onset due to thrombosis, and called acute bulbar paralysis; here the reflex action is lost, and there is loss to

faradism, while sensation may or may not be affected. In cases where the nucleus itself is affected, as a rule other muscles which are supplied by the accessory to the vagus, namely, the muscles of the vocal cords and larynx generally are implicated; and very often the muscles supplied by the spinal accessory, viz. the sternomastoid and trapezius. Then, with the gradual onset of progressive muscular atrophy, the tongue is liable to become affected as well as the vocal cords. When one comes higher up to the motor tract, just above the nucleus, the paralysis will be unilateral, and the limbs probably will be affected on the opposite side to the paralysis of the palate, also the lower part of the face and the tongue.

In our first patient the lesion is between the decussation and the cortex, or in the cortex. In many cases it is of sudden onset, and is due to thrombosis of the vessels which go into the medulla and the pons; or possibly it is higher up still, in the internal capsule or in the cortex. As a rule, in such cases the thrombosis is unilateral, the exception being where there are two attacks, one on each side, producing paralysis of both sides of the palate. That, I believe, is the condition of the woman who has not attended, and whose notes I read you. You will remember she has absolute loss of movement of the soft palate on either side, but has movement to reflex irritation; therefore the lesion must be higher up than the nucleus of the accessory to the vagus. From her history I have no doubt she has had a lesion on both sides; on the first occasion it was on the left side, on the next it was on the right. She also lost speech completely, and it has been shown by Dr. Semon and Mr. Horsley that if phonation is absolutely lost, there must be a lesion on both sides to affect both internal capsules. Probably the lesion was not in the cortex, as she had no convulsive fits. Her case, indeed, is what would be known as pseudo-bulbar paralysis. It shows all the symptoms of bulbar paralysis, like the man who had amyotrophic lateral sclerosis, but differs from it in the suddenness of its onset and in the muscles not having lost their electrical reactions nor their reflex response.

Now let us consider the diagnosis of the disease in the man I have shown you, who has hard, enlarged glands. You will remember the onset

was gradual. He has paralysis of the soft palate on both sides, and paralysis of the fifth nerve on one side. He has also lost taste completely on one side. In the first place, there is no movement of the soft palate on phonation, and no reflex movement when it is tickled; therefore the lesion must be in the nerves, or in the nucleus of the accessory to the vagus. Besides that, he has paralysis of the fifth nerve on the right side and loss of taste over the whole of the right half of the tongue, a condition which has been observed in lesions of the root of the fifth nerve. The question is whether it is possible for the symptoms to be caused by one lesion. I do not think it probable; I think there are two lesions. The vocal cords are in good order according to Dr. Semon, the sternomastoid and trapezius are not affected, and we have to ask ourselves whether it is due to a malignant growth. Syphilis seems out of the question, and Dr. Semon suggests the possibility of a lympho-sarcoma. The lesion must be either in the bulb or in the nerves coming from it, on account of the loss of reflex action and the reaction of degeneration. Is it in the grey matter or in the nerve? Against it being within the medulla is the fact that the vocal cords are intact. Is it in the pharyngeal plexus? Is there a growth there which is causing the enlarged glands? Or is it a meningitis? I think the evidence, on the whole, points to the lesion being outside the medulla, and affecting the pharyngeal plexus.

Next arises the question as to the importance of diagnosis in these cases from a prognostic point of view. It may be difficult to decide whether the disease is in the nucleus, in the pyramidal tract, in the internal capsule, or in the cortex; but it is important to do so, because of the different prognosis. Two cases may at first sight appear quite similar, but in their histories may differ widely. Moreover in one there may be no wasting at all, and no lost reflex action, while in the other there may be marked wasting and changes to electrical currents.

To sum up, the prognosis in diphtheritic paralysis is favourable, in progressive muscular atrophy, or in amyotrophic lateral sclerosis it is very unfavourable, as the disease will steadily progress and probably cause death in a year or two; while in acute bulbar paralysis and in acute

lesions of the nucleus, or of the motor tract above this, or in the internal capsule or cortex the prognosis is better, as the patient may improve ; and even if he does not, the disease will not advance, except he have another attack, to which he may succumb. Hence, excepting diphtheritic cases, all paralyses of the soft palate are very serious.

THE PRACTICAL USES OF HYPNOTISM IN PUBLIC CLINICS.

BY

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FOR several years past in the Neurological Clinic at the Post-Graduate Medical School, an occasional patient has been treated by hypnotic suggestion ; the work was rather unsystematically done, and records were not kept with sufficient completeness to be of much value for reference. During the last winter a much larger number of patients have been so treated, and the method of work has been more systematised. A room has been set aside, called "the sleep room," for carrying on this special line of treatment ; full records have been made of the cases treated therein, and the results of treatment in each case have been carefully noted.

It will be interesting and profitable, I believe, to report some of the results obtained in this work.

At once I may say that my experience in treating a certain line of diseases has been such that the prejudice first felt in employing this method, a prejudice quite prevalent among the profession in this country, has been entirely removed, and I fully believe in the benefits to be derived by hypnotic suggestion properly employed. Hypnotism has great value in treating certain diseases, though its place in therapeutics is peculiar. My feeling is that its value is more striking in its effects than wide in applicability.

There are many conditions, mental, moral, and physical, to be met among the sick, and to be combated by one method or another ; these conditions, some of them, cannot be met by the ordinary therapeutical resources, and they make the successful management of a case oftentimes most perplexing. Any agent meeting the require-

ments of these cases will be welcome to the physician, and as much a relief to him as to the patient.

The physicians attending the clinics at the Post-Graduate, and who may be taken as reasonably representing the attitude of the profession at large, have evinced an active interest in this method of treatment, but, at the same time, have also displayed only a slight understanding of it, both as regards the method of employing it and the benefits to be derived from it.

I hope, then, to indicate in this brief article some of the diseases for which hypnotism may be used ; to explain our manner of employing it, and to remove to some extent the attitude of mild disfavour with which we feel it to be regarded by the profession at large.

The method I employ for inducing the hypnotic sleep, is the one in most general use, and is commonly referred to as "Bernheim's method;" it is very simple. In the first place, it is so arranged, as far as possible, that every condition will favour the development of the thought we wish should dominate the patient, namely, that he is to go to sleep. To this end, then, must be considered the mental state of the patient, the attitude of the operator, and the environment. It is my opinion that revolving discs, or any methods of seeming mystery, employed for the purpose of inducing the hypnotic state, are an imposition on the patient. I prefer to influence him by appealing only to his confidence and intelligence.

It is explained, then, to the patient that, for the proper treatment of his disease, he is to be hypnotised ; that the treatment, it is believed, will be effectual in its results, and that certainly there is nothing unpleasant in the employment of it. His nervousness, and any scruples or fears, are overcome by a reasonable explanation of our object and method, or by any means suitable to individual cases. We aim to secure his entire confidence and to place him at ease.

The attitude of the operator only need be quiet, confident, and firm ; any nervousness, or agitation, or uncertainty on his part, is communicated immediately to the subject.

There should be no noise in the room, and it is probably more favorable to have the room somewhat darkened.

Then the patient is placed in a chair sufficiently

easy to produce no physical constraint ; his head rests against the back of the chair or against the wall, he is directed to fix his eyes upon some object which is held up before his face, or upon any object in the room. And so, having the patient in a mental and physical condition favorable to the hypnotic state, he is told quietly, firmly and repeatedly, to "go to sleep." His brow is usually stroked. In a short time his eyes look sleepy, eyelids become heavy and tremulous ; he is then commanded to close his eyes, the hand of the operator is passed gently over the eyelids, and the patient is told he cannot open his eyes until told to do so. In almost every case the patient will be in the first stage of hypnotic sleep.

With some, and especially with those who have had one or more treatments, the simple command "Go to sleep !" is sufficient. But in others it may be difficult to develop the state, and the method for inducing it must be persevered in for some minutes ; however, if, after ten minutes of patient, persistent effort, the subject indicates no degree of somnolence, no further attempt to hypnotise him is made that day. Next time, possibly, the patient, feeling less nervousness and more composure, passes quickly and easily into the hypnotic state.

At the Post-Graduate it is not always possible to shut all noise out from the room, and there may be a number of students in to observe the operator at his work. These conditions are somewhat unfavorable to success and should not exist ordinarily, as, especially in new cases, they directly oppose the influence necessary to carry out the treatment.

No experimenting with the patient, simply to gratify the curiosity of the spectators, or the operator, is ever done. It is manifestly unfair to the patient, and undignified, to take advantage of his passive state for the amusement of spectators or the gratification of curiosity. The treatment of his disease is the only reason for putting the patient into this state.

It has been my observation that an effort on the part of the patient to further the efforts of the operator, an effort to go to sleep, may really defeat the development of the condition. The mind of the patient must be absolutely passive.

The degree of hypnotism need never, as a rule, exceed the first stage, the somnambulic state ; though it will be found that some patients,

especially susceptible, will pass so readily into the further stages that even the stage of rigidity may develop with no desire on the part of the operator to induce it.

All that is desired, all that is necessary, is to have the mental state of the patient plastic and receptive to whatever suggestions are to be made to him.

We find it best to make only one or two suggestions, repeated over and over several times, at one *séance*. At the next treatment another suggestion is added to and repeated, with the first one made. In this way the condition of the patient is gradually corrected.

The duration of sleep is fixed by the conditions for which treatment is being given, and varies according to the indications in individual cases ; in some conditions ten minutes is enough, in others half an hour, or more, is given. Observations in this matter of time have shown that the longer period for sleep is of advantage in many cases. It seems that in this way suggestions are better received and retained ; and many cases of irritable nerves and worried brains are noticeably soothed and refreshed by the prolonged sleep.

To end the sleep, the patient is simply commanded to wake up.

The forms of disease in which we have found suggestive treatment most useful are, naturally, those of a functional nervous nature. And if we find its value indicated in no other lines of disease, its value here is often most marked. Primary types of neurasthenia and major types of hysteria furnish the best cases for the employment of hypnotism. Insomnia, when associated with these morbid states, is sometimes relieved by a single *séance*.

Organic diseases have sometimes associated with them, intensifying and multiplying their symptoms, some functional trouble, which may be removed by this method of treatment.

Phobomaniacs. — Persons suffering from fixed ideas and morbid fears are favorable subjects for suggestive treatment. Among those successfully treated in the sleep room were : A young woman with agoraphobia, two male dipsomaniacs, also a tobacco habitué (man), a young man with syphilophobia, another with *folie génitale*, several with persecutory ideas, numerous neurasthenics, and several hysterics.

The peculiar mental foibles of a paranoiac, the

idée fixe of a melancholic or a neurasthenic, the perversities of a degenerate, the vagaries of a hysterical, and the instability of thought and purpose often characteristic of many of these cases, may show better results under this method of treatment than under any other. Certainly it is accomplishing a great deal for these patients to make definite and consistent and consecutive habits of thinking and living take the place of purposeless and aimless ones ; to implant in an enervated will resolution enough to overcome some vicious habit hateful to the individual, and which is sapping his moral energies ; to enable the individual who has lost the command of his native resources to regain his bearings and place himself in his normal social condition.

Statistics of results.—Our records show thirty patients to have been treated in the sleep room from the 1st of October, 1895, to the 1st of March, 1896. Of this number two only could not be hypnotised, and three others were brought under the influence only after several attempts. All the others (twenty-five) were easily rendered passive, passing promptly into the somnolent state, and some becoming cataleptic.

It can be stated that where the method could be employed, it in no case was found entirely useless ; to some degree it did good in every case, and in at least half of the cases its benefits were most marked.

The appended histories are given to show the class of cases selected for this treatment. They are chosen as illustrating characteristic but not striking cases :

Case 1. Dipsomania.—J. J., æt. 36, United States, journeyman. This man is employed in the folding room of one of the large newspapers of this city. He had been addicted to the excessive use of alcohol for ten years. He presented himself at our clinic in October, 1895, suffering with acute alcoholism, and was on the verge of delirium tremens. Our usual treatment for this condition was given him, and he was, of course, instructed to stop his drinking. He did restrain his inclination for drink and was progressing favorably, when one day, about the tenth of treatment, he returned in as bad condition as on the first. He had been unable to repress the old habit any longer. It then occurred to us to hypnotise him and treat the habit.

The first attempt to so treat him was not successful, as, owing to the effects of alcohol, he was in an excited state. The second attempt was successful, and he was hypnotised twice a week for a period of eight weeks. While in the hypnotic state it was suggested to him that he would not drink any more ; that he would despise all forms of intoxicating drinks. The treatment resulted in a cure of the habit.

Remarks.—In January, 1896, this patient was in Boston for two weeks ; while there business matters annoyed him somewhat, and he drank one or two glasses of whisky one evening. This is all he has drunk since the first *séance*. He is still under a general tonic treatment, as his long-continued habit has left him somewhat neurasthenic ; and he is hypnotised once a week, though he states that he has not the slightest desire to drink.

Case 2. Agoraphobia.—Mamie M., æt. 19, United States. Patient had always had good health until two years ago, when she developed tertian type of intermittent fever. Under treatment patient recovered from this trouble, but began to manifest peculiar nervous symptoms. She became excitable at times, and complained of vertical pain and pressure, and of an indefinable feeling of fright ; then developed a fear at being left alone. She could not be alone either in the house or in the street ; the thought of being so filled her with horror and agitation ; the vertical pressure would become intensified ; vertigo attacked her, and a sensation as of some one pushing against her back.

This condition had existed for eighteen months. Her condition under hypnotic suggestion was improved.

Remarks.—The patient lived in a remote part of Brooklyn, and some other member of her family had always to come with her to the clinic ; this was inconvenient, and treatment was discontinued before recovery was complete. But she was under our care for three weeks, receiving two treatments per week, and improved to the extent of being able to be alone in the house or to go a distance of three or four blocks from her home alone.

Case 3. Neurasthenia of adolescence.—E. F. J., æt. 23, United States, stenographer. This young man had practised onanism from onset of puberty to about his eighteenth year. Graduated from public school when eighteen years of age. Was a stenographer after that until about his twenty-first

year; then writer's cramp, which had been developing during a period of six months, affected his right hand so severely that he had to abandon his work. Recovered free use of hand. *Folie génitale.*

Patient, at the time of presenting himself at the clinic, had been unable to give any attention to his business for two years; was unable to read, or write, or talk to other members of family or to friends, because of an intense and peculiar excitement to which he was subject. These spells of excitement would come on whenever the patient made any effort of any kind, mental or physical, and were characterised by extreme mental excitement and perturbation, by vertigo, a feeling of distress, and general tremor. These attacks had come to dominate him to such an extent that the patient had gradually drifted into a condition of social isolation and absolute inability to apply himself to any line of work. His individual adjustment to environments had been lost, and although the young man is intelligent and possesses average moral force he could not find his place in the business or social world.

He had been under various treatments during these two years, but with entirely negative results. He was unable to follow any hygienic instructions as to bathing, exercising, etc., because these spells would interfere with whatever he undertook to do.

We gave him very little medicine, but he was hypnotised, and it was suggested to him that he would be able to ride a bicycle for one hour every day; that he would walk two miles every day, take a cold sponge bath every morning, and read so many minutes every morning and evening; that these spells would not interfere with this prescribed course of habits, and that, indeed, by applying himself to these things he would find himself less subject to the spells. He began to improve at once; resumed his studies, followed with no trouble the things suggested to him to do, and again interested himself in those around him. In two months' time, with ten treatments, the patient was completely cured, and he went to work.

Remarks.—Being employed in a large furniture house where his work during the holiday trade became very heavy, he felt at one time a threatened return of his trouble. He was hypnotised once, and had no further inconvenience.

This case illustrated most strikingly how an in-

dividual with intelligence quite up to the average, and moral force equal to those in his own conditions of life, can become useless to himself and to society. It also illustrates the value of hypnotic suggestion in such cases.

Case 4. Traumatic hysteria.—L. K., æt. 50, United States, merchant. This man had been a heavy smoker from very early years. His health, he states, had always been good until two years ago. At this time he accidentally fell in the street. The fall was not severe, and did not disable him particularly; but soon afterwards there began to appear certain nervous symptoms. The patient became emotional and excitable, would cry easily, and was often greatly depressed; could not apply himself to business. Sight and hearing became impaired. Finally he would make no attempt to read or write, as he declared that using his eyes produced a severe pain in them, and in his head. Careful examination of the eyes revealed no condition not met by the glasses he had been wearing, and no form of organic trouble was discovered.

There is no doubt but that the tobacco habit had prepared this patient's nervous system for the development of a functional disease, and the slight injury or shock of the fall was sufficient to inaugurate the hysterical condition.

He was told to read, and would try to do so, but always stopped in a minute or two, put his hand to his eyes, and protested that he could do no more as the pain was so intense. He was told to stop his habit of smoking, and he tried to do that, but finally declared he was not strong enough to stop.

He was placed under hypnotic treatment. He was very easily hypnotised, and while in this state he was told that he would now be able to read as soon as he awoke, and that he would not smoke any more because he did not care to smoke. He was able to read, as told, stopped smoking without any trouble, and improved markedly in general health.

A woman with hysterical contracture of the right arm, who had not been able to use the arm for many months, was made well in one treatment.

Another patient, a man of forty-five, a marble-cutter, sustained a severe blow upon his chin, and presented himself at the clinic three months after this accident, a well-marked case of traumatic neurosis. He complained of severe pains radiat-

ing from shoulders to vertex of head; he was emotional and intensely excitable, and mentally depressed. Held his head in rigid condition. He was hypnotised, and it was suggested to him that when he awoke he would have no pain and would be able to move his head. One *séance* effected a complete cure of these symptoms.

Conclusions.—These cases illustrate sufficiently the class of disease in which we find hypnotic suggestion useful.

We repeat that this method of treatment is of value in cases of habit and neurasthenic insomnia, major types of hysteria and allied diseases; that it is of great service in treating the various morbid moral conditions in which we all find so many of our patients. But while it can serve so beneficially in the management of such cases, it is, of course, unreasonable to expect to implant in a paranoiac or a degenerate, characteristics forming no part of his original mental and moral endowments.

The Post-Graduate, July, 1896.

THERAPEUTICAL NOTES, &c.

The Celluloid Mull Bandage.—It has been found that celluloid will dissolve in acetone into a thick gelatine, which can be used for casts, as it hardens sufficiently in an hour and a half, and becomes absolutely solid in three to four hours. The sheet of celluloid is cut with a pair of scissors into scraps and placed in a wide-mouthed bottle, filling it a quarter full. The rest of the bottle is then filled with the acetone, and the contents stirred with a stick occasionally. When rubbed on the mull bandage it forms the cleanest, hardest, and by far the lightest substance known for this purpose, while it is not affected by the secretions of the body. It has been used at Strasburg with great success, and is warmly recommended as a most valuable bandage on account of its cheapness, durability, solidity, and elasticity, especially for use in polyclinics. It is not necessary to have as many layers of the bandage as with other substances, and the celluloid rubbed in for the outer layer forms a handsome finish. As it is very sticky and can only be dissolved with acetone, it is best to wear leather gloves while handling it. It is especially adapted for permanent removable

casts, with or without splints. In some cases a cast or model of the limb or part has to be made, and the celluloid cast modelled on this.—*Cbl. f. Chir.*

Bekarewitsch Treatment of Varicose Ulcers.—After the spot is thoroughly disinfected, a piece of gauze smeared with a 10 per cent. boric vaselin is laid over it, through which it is delicately massaged for five to ten minutes, and then more energetically. It is then dried and dusted with iodoform and wrapped in an anti-septic bandage covered with the following:—Zinc oxide and gelatine, equal parts; glycerine and distilled water, four times the quantity, equal parts. The whole is then enclosed in a starch bandage and left undisturbed for two or three days, during which time the patient can go about and do light work.—*Therap. Woch.*

Improved Method of Narcosis.—Gräfe has found that the sensitiveness of the nasal membrane is of far more importance in terminating narcosis than is generally supposed, owing to its reflex action on the vagus region. If the nose can be kept closed, the narcosis proceeds far more readily and rapidly, and lasts much longer, with less of the anaesthetic required. He has therefore invented a light spring pad with which he stops the nostrils before the operation, and does not remove it until the patient is completely aroused. It has rendered surprising service in the narcosis of persons with pronounced heart troubles, with the minimum of after effects.—*Cbl. f. Chir.*

Prevention of Gonorrhœa.—Von Neisser has theoretically confirmed the method of preventing gonorrhœa advocated by Blokusewski-Daum. This method consists in dropping into the meatus, the lips of which are held open, two drops of a 10 per cent. solution of nitrate of silver. One drop is allowed to run over the frenum. Fifty patients adopted this treatment without experiencing the least irritation. Five seconds' application of this solution always inhibited the growth of the gonococcus when cultivated in artificial media. Weaker preparations are unreliable.—*Centralblatt für die Krankh. der hard und sexual Organe.*

THE CLINICAL JOURNAL.

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A CLINICAL LECTURE ON DISEASES OF THE TONGUE.

Delivered at St. Thomas's Hospital

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GENTLEMEN.—The man before you is 58 years of age, and is the subject of old syphilitic disease of the tongue. You can see that he has a considerable amount of deep fissuring of the tongue, and in one place it is superficially ulcerated. The ulceration is supposed to be in a condition of incipient epitheliomatous disease; but there is nothing very marked about it. The history of syphilis is quite clear, and the condition of the tongue is characteristic of syphilitic invasion in the tertiary period.

This other patient, whose age is 54, has a raised ulcerating patch on the tongue, with exceedingly well-marked induration at the base. In view of the fact that his glands are also enlarged and hard under the jaw, and that the margins of the sore are infiltrated, it seems to me that an epitheliomatous change has already commenced in the base of the ulcer, and that the disease will prove on examination to be of an epithelial type. In this, as in any other doubtful case, one can assure oneself by snipping off a small portion and examining it microscopically.

The syphilitic patient has improved very much under anti-syphilitic treatment, and although the occurrence of epithelioma is suspected, I do not think there is sufficient evidence to justify one in supposing that the disease has as yet become of an epithelial character. These syphilitic tongues of very long standing, of which this is an example, are apt in many cases to assume eventually the characters of epitheliomatous disease.

With reference to diseases of the tongue generally, if dealt with in a systematic fashion, one would of course refer to those somewhat rare cases of congenital defect. One instance has been recorded in which the tongue was altogether absent, being only represented by a small papilla in the floor of the mouth. This condition is so rare that only that one instance has been mentioned. Far less infrequently the tongue presents a cleft appearance—either in the middle line, when it produces a bifid tongue, or as a double cleft, which will cause it to be trilobed. We meet with cases where there is a short frenum, which mothers are apt to regard as a serious trouble; it is commonly called tongue-tie. In these cases I warn you not to yield too readily to a request for division of the frenum, because, as a matter of fact, it is very rarely (as many suppose it to be) a cause of deficient power of articulation. Very often the fact that the child is slow in learning to speak is due to other causes. In some cases, however, the frenum, which is naturally very distensile, and readily stretches when the tongue is used in speech and otherwise, becomes toughened and thickened by inflammatory changes of a chronic character. In these cases it may be desirable to nick the frenum slightly, taking care not to do too much, or you will enter the muscular tissue and loosen the anterior connections of the tongue. Cases are known in which such a course has caused suffocation by the tongue being drawn back after the too free incision. Petit, I believe, has described one or two cases in which an unnaturally long frenum has permitted the so-called swallowing of the tongue, causing suffocation. This may be prevented, of course, by pulling the tongue forward.

Another congenital condition sometimes met with in children is a very great hypertrophy of the tongue. It projects out of the mouth, interfering with the functions of the organ. It becomes congested by pressure of the teeth, roughened and indurated on the surface, and is associated with very considerable salivation. The pressure of the enlarged organ on the teeth causes, in time, marked displacement, especially of those in the

lower jaw, which may become almost or even quite horizontal. The exact nature of this condition of macroglossia, as it is called, was unknown for a considerable time, but it really is, as Virchow has pointed out, essentially a lymphatic distension and infiltration; white cells are met with in great abundance in a delicate stroma, forming a kind of lymphoid tissue, which is developed to the greatest degree on the deeper aspect of the tongue, while the connective tissue of the organ is increased throughout. For this there is no treatment except excision. The end of the organ may be cut straight across, when the cut surface shrinks and the tongue also; or a V-shaped portion can be removed, and the two surfaces united together.

I have here admirable drawings of what I hoped to be able to show you in the living subject. The tongue presents a whitened surface, which in some cases may be patchy, in others more uniformly covered, with red spaces intervening. This condition is known as chronic superficial glossitis, ichthyosis linguae, psoriasis of the tongue, or smoker's tongue. The latter name was given to it in consequence of the frequency with which excessive smoking is associated with its occurrence. As its name implies, it is essentially a very chronic superficial inflammation of the tongue, without, as a rule, any ulceration. The cause is local persistent irritation. Over certain areas the tongue will be seen congested, and devoid of the white investment which is seen in other parts. At first the papillæ become hypertrophied, and afterwards, as a result of the chronic inflammatory changes which take place in them, they become atrophied and disappear; so that in certain congested areas one finds a surface of a smooth, glossy appearance, devoid of the papillary layer, which has been destroyed. In some places the epithelium is shed, and in other parts it accumulates, and forms a thick, very intimately adherent layer of whitish grey or yellowish white epithelium of varying thickness. The healthy papillary surface is gone, being replaced here and there by bald patches, at others by heaped-up leucomata; elsewhere it may show cicatrices of former ulceration. When the disease is met with at an early period, appropriate treatment will result in cure; but in many instances the patient suffers so little inconvenience that relief is not sought for until the condition is very advanced. After the disease has existed

many years, treatment is very unsatisfactory. Of course, the cause of the disease must, if possible, be removed, and any irritant action stopped, but even then the disease is often inveterate and very difficult to deal with. In some cases a gargle of 20 grs. to the ounce of bicarbonate of soda is useful, and in others the patient should frequently wash out the mouth with chlorate of potash, 5 grs. to the ounce of water. These measures may, at all events, stay the progress of the disease. In early cases one notices an erythematous border round the patches, which indicates that the disease is advancing. In others, where this state of the tongue has existed for ten, twenty, or thirty years, there are no inflammatory appearances, and the red border I have spoken of does not exist.

The great importance of this disease is not in the symptoms, or the distress which it causes to the patient, so much as in the fact that it is a strong predisposing cause of cancer of the tongue; the leukoplakia becomes warty and then epitheliomatous, or a bald patch begins to ulcerate and takes on the same action. It has been estimated that as many as one third of the cases of cancer of the tongue have had this as an antecedent condition. The glossitis may have existed a very long time before any carcinomatous change occurs, but in all these cases you must be on the look-out for the possible occurrence, sooner or later, of ulceration and induration, which will suggest an ingrowth of the epithelium into the muscular substance of the tongue, and the consequent transformation of innocent into malignant growth. When this takes place, as evidenced by the appearances I have mentioned, the removal of the organ will be indicated. In doubtful cases it will be well to cut away and examine a portion of the diseased area under the microscope.

I shall only say a word or two about ulcerations of the tongue. There is a simple ulcer, which may result either from the irritation of a tooth, generally placed on the border of the tongue, and usually in the posterior half of that border, because it is there it comes into contact with the teeth. This form of ulcer is generally round in form, with somewhat abrupt margins. It forms an irregular depression on the margin of the tongue, with an inflamed area around it. The surface is suppurating more or less, and grey in colour. It is accompanied by a sharp, cutting sort of pain. An injury of the tongue,

such as a bite, or the continued presence of the rough end of a clay pipe, may cause a similar condition. The cause of the irritation should, naturally, be removed; if the source be the roughened edges of teeth, they should either be made smooth or extracted. Ulcerations produced by mercurial poisoning—mercurial stomatitis—are not very dissimilar to those I have described. They are superficial, irregular ulcers, especially on the under surface of the tongue; they are also found inside the cheeks. In cases of mercurial poisoning there is salivation, and the disagreeable mercurial fœtor. One does not often see cases of this kind now, because it is recognised that salivation, which formerly was regarded as an index of the therapeutic influence that the mercury was causing, is really a poisonous effect, to be avoided by every possible care.

Tuberculous ulceration of the tongue is an exceedingly rare affection, and generally occurs when tubercle is present elsewhere; it does not occur primarily in the tongue. It is usually placed far back on the dorsum; the edges are infiltrated in places with inflammatory products, but not hard—usually slightly undermined. The ulcers are small, round or oval shape, about the size of a linseed, chronic, and not very painful. When they do occur on the tongue, the patient has usually advanced phthisis.

There are other ulcerating affections of the tongue which I need scarcely trouble you with, such as that produced by actinomycosis. Then there is the aphthous ulcer associated with the irritation of the gastro-intestinal tract so common in children. As a result of some of these ulcers, the tongue may be bound down by adhesions to the floor of the mouth, and its functions are greatly interfered with.

A more common and much more important ulceration of the tongue is that associated with syphilis. Syphilis of the tongue may be congenital, appearing either as a manifestation of secondary syphilis or a gumma, and not presenting any difference in type from similar conditions occurring in later years as the result of acquired disease. Apart from congenital cases, syphilis may affect the tongue in either the primary, secondary, or tertiary stages. When you see a young man or woman with an ulcer on the tip of the tongue, presenting a raised base, associated

with marked induration, having a shallow, depressed surface, not inflamed, nor suppurating, nor painful, with well-marked enlarged glands under the jaw, you should remember that these conditions strongly suggest that the patient is suffering from an infecting chancre of the tongue, and its progress and treatment will not differ from that of the same condition in any other part of the body. In the secondary stage of syphilis there are the characteristic mucous patches, which consist of an area of infiltrated submucous tissue, covered with an abundant epithelium on the surface. They are generally oval or circular in shape, greyish in colour from maceration of the epidermis, and occur some three months after infection. They are met with not only on the tongue, but on the insides of the cheeks and on the fauces, and are often associated with condylomata around the anal orifice, and with signs of secondary syphilis elsewhere. The treatment is that calculated to relieve this stage of the disease—the internal administration of mercury and its local application as a gargle seem to have an excellent curative effect.

In the tertiary period of the disease there is gummatous infiltration, either in the single or multiple form. Numerous gummatous masses may be met with; they may occur in any part, but generally in the middle portion. The lesions develop in the muscular and fibrous tissue, and form masses which at first are of considerable hardness. Subsequently they increase in size, become softer, presenting finally a soft, more or less elastic, painless swelling, covered by smooth and generally reddened mucous membrane. Even in this liquified condition gummata become absorbed under treatment, and their traces may almost wholly disappear. In other cases, where suitable treatment has not been resorted to, the mucous membrane gives way, the contents escape, and an irregular cavity is left with a sloughy surface of yellowish hue, extremely adherent to the tissues underneath, from which it cannot be scraped off or removed. The appearance has been called, not very strictly, "wash-leather slough." There is no considerable induration of the surrounding parts; there may be some inflammatory hardness, but nothing of a special character. The edges of the ulcer are very thin and undermined, which is an important point. Little or no pain is associated

with the condition, and the lymphatic glands under the jaw are not affected. A history of syphilis may be obtained, and possibly there are traces of the disease elsewhere on the body. Then, as a result of long-continued disease which has been treated intermittently, there is the chronic infiltrated condition of the tongue I showed you in the first patient, with numerous fissures and a hardened nodular surface, a state which cannot be affected readily by treatment. This is, probably, also a pre-cancerous state, or one in which cancer is very prone to supervene.

We may now consider conditions of the tongue associated with new formations or growths. These may be either simple or malignant, solid or cystic. The cysts which sometimes develop in the tongue are usually retention-cysts, either in the substance of the tongue itself or underneath it in connection with the mucous glands, found abundantly on the under surface. A duct may get obliterated, and as a result a tense, elastic tumour forms, with the mucous membrane tightly stretched over it so as to render the swelling semi-translucent. The name ranula has been given to it, because the colour resembles that of the belly of a frog. The disorder is generally more to one side of the middle line than the other, and was formerly believed to be associated with obstruction of Wharton's duct. That is easy to disprove by seeing the saliva flow out of the duct, by being able to pass a probe along the submaxillary gland, and by absence of any alteration in the consistence of the gland itself during mastication. When a calculus obstructs Wharton's duct, one finds that during the period of mastication the gland becomes very hard and painful from the accumulation of saliva behind the obstruction, and the calculus can often be felt in the floor of the mouth. None of these symptoms occur in the case of a simple retention-cyst due to obstruction affecting the sublingual glands. The disorder is generally disposed of by removing a portion of the cyst-wall, in sufficient quantity to prevent any chance of the opening becoming closed; the lining membrane may be cauterised with advantage, a mere puncture and evacuation of the contents is useless. The shrivelling up and disappearance of the cyst cavity is not a uniform result; some cases give a good deal of trouble, but the majority yield to excision of a portion of the cyst wall.

There is another form of cyst met with in this situation which can be felt in the floor of the mouth under the tongue, and sometimes in the neck. I refer to the congenital or dermoid cyst. The mucous membrane is not adherent, and is, therefore, freely movable over the cyst, in which respect it differs from a ranula. The resemblances and differences are parallel with those observed in ordinary dermoid and sebaceous cysts of the head. Dermoids at the outer margin of the orbit are found to be more deeply placed; the whole of the soft tissues cover the tumour, which adheres closely to the bone and pericranium. Dermoids are not so tense as sebaceous cysts, which are in immediate connection with the skin, and quite superficial. Again, dermoids are more doughy in consistence than fluctuating; they are caused by some portion of the epiblast being infolded and shut off from any communication externally. It thus forms a sac wherein the secretions of the skin accumulate. The sublingual variety may occur between the two genio-hyglossus muscles, when the growth will be central; or it forms between the genio-hyglossus and the mylo-hyoid, when it is one-sided. It often has extensions in various directions, and these deep connections have to be considered and dealt with in any attempt at surgical treatment. As I have said, it generally presents beneath the jaw, and fluctuation or semi-fluctuation can be felt with one finger in the mouth and one on the skin of the part of the neck adjacent. I remember a case in this hospital which was somewhat obscure; it occurred in a young woman. A puncture was made, and a trocar driven into it; but instead of the clear fluid which was expected, epithelial débris and sebaceous matter came away. Considerable inflammation followed. The neck became swollen and oedematous, respiration was soon obstructed, and the patient nearly died; a tracheotomy saved her life. I mention this to show the dangers of mere puncture of these cysts. The proper surgical treatment is to dissect out and enucleate the cyst, with its contents, though in doing so you may find some embarrassment in the fact that they may extend very deeply into the neck, with attachments to the hyoid bone and the styloid process and in unpleasantly close relation to some of the important structures in this situation. However, you must endeavour to enucleate the cyst.

completely: if you leave any portion of its wall behind, the disease will recur.

A papilloma or warty growth in the tongue is not uncommon; of this I am able to show you some specimens. One of these was a case of my own, in which there is a warty development in the middle line at the back of the tongue. You will see that it is covered with sodden epithelium, which somewhat masks its appearance. These papillomata occur at any period of life; they are common in persons the subject of chronic glossitis. In the young they are harmless, but in persons of 40 or 50 years of age you must always bear in mind the possibility of epitheliomatous transformation, and this is one of the ways in which an epithelioma very commonly begins. I believe the patient from whom this specimen was taken had a warty growth for many years, but after a long interval the tongue had to be removed on account of commencing epithelioma. Even where there is no evidence of this, the warty growth should be excised, taking the adjacent portion of the tongue along with it.

Tumours of the tongue of a simple character, such as lipomata, fibromata, and enchondromata, are exceedingly rare. Of malignant tumours the same may be said with regard to sarcoma as a primary growth in the tongue; probably primary sarcoma does not take place there. Some cases have been detailed, but it is doubtful if they have been correctly described. Of course sarcoma may develop in the tongue as a secondary formation from a sarcoma in some other organ. With regard to carcinoma, that, unfortunately, is exceedingly frequent in the tongue, and I thought it better to deal with this part of the subject in a separate lecture.

very offensive discharge from the right ear for about nine months, concurrently with an attack of prolonged digestive disturbance. It was a well-nourished child, of healthy parents, and having two particularly healthy brothers. It had suffered greatly from pain in the ear, and there were enlarged glands in the neck, probably tubercular in nature. I saw the child in November, 1895, and the ordinary treatment by cleansing and antiseptics was regularly carried out, with little or no benefit. The membrane was then extensively destroyed, but there were no granulations visible. Dr. Murray, of Beckenham, under whose care the child was, rightly conjectured that necrosis existed on account of the profuse and very foetid nature of the discharge. In January, 1896, some tenderness and swelling was observed over the mastoid, but these signs were not at all well marked. On January 24th the following operation was performed, Dr. Murray assisting and Dr. Bolas administering chloroform. The auricle was turned forwards by a semilunar flap, and a small sinus which existed was freely enlarged with gouge and mallet until the necrosed cells were opened. Much débris was evacuated, and then at a considerable depth a rough sequestrum of irregular shape, about the size of a small hazel-nut, was felt and with some difficulty extracted.

The facial nerve was apparently not implicated. The cavity was well cleansed and stuffed with iodoform gauze. The external wound behind the ear was left quite open, some of the integument being cut away over the opening in the bone and a portion being tucked into the cavity. In the after treatment of the case the opening was persistently kept patent, and the cavity well washed out with antiseptics. The child lost its pain and gradually improved, but the sinus was not allowed to close until May, the discharge, which had been very slight ever since the operation, having entirely ceased. The present condition of the child is satisfactory. There is no discharge from the canal, and the wound has closed. The glandular enlargement in the neck renders residence at the sea-side desirable. On April 4th one of these glands, having broken down, was opened and scraped. Much of the success in this case was due to the persistence of Dr. Murray in keeping the wound behind the auricle open. The sense of hearing will, of course, be permanently destroyed.

CLINICAL NOTES

FROM

A. MARMADUKE SHEILD, M.B., F.R.C.S.

Mastoid Disease (Tubercular) in a Child aged 12 Months—Removal of a deeply seated Sequestrum—Cure.

FOR the notes of this case I am indebted to Dr. Murray.

A child, æt. 12 months, had a purulent and

Remarks.—Mastoid disease associated with the formation of sequestra are the most favourable for operation. In my experience such sequestra are far more common in children than in adults. In delicate children, especially after scarlet fever, considerable portions of the temporal bone may separate and be removed, and it is quite extraordinary how such important structures as the facial nerve or the carotid vessels seem to escape injury. In addition to the important signs of excessive foetor and discharge, the sequestrum may be felt with a probe, and swelling and the formation of abscess behind the mastoid is an important indication for operation. I feel sure that the most certain method of getting cases of mastoid disease to heal soundly is to remove the bone very freely, and actually to cut away the soft parts behind the ear, to prevent closure tucking down a flap of skin into the cavity. The large gap in the cavity thus made is stuffed daily, and the medical man had better be quite indefinite as to the time such a wound should be kept open. My own rule is to keep such cavities patent as long as this can be accomplished. Nine months is no uncommon period. Of all local applications, I think there are none superior to a 10-volume solution of hydrogen peroxide, which cleanses the intricate depths of these wounds in a most remarkable manner. If added to all this treatment be good diet and sea air, the most desperate cases will ultimately heal and do well. In children of the tubercular diathesis many of these cases are undoubtedly really tuberculous caries or necrosis. Some of them are due to congenital syphilis, and will heal in the most rapid manner under the appropriate remedies for this condition. Finally, it may be added that it is well not to operate in these cases unless there is a reasonable probability that the sequestrum is loose. It often requires all the tact and management of a medical man to delay operative proceedings in cases associated with pain and discharge. If operation be undertaken before the diseased bone has loosened, some of it is very apt to be left behind, and the pulling away forcibly portions of bone in the neighbourhood of the vessels or facial nerve is apt to be followed by disaster.

Cases of Exostosis.

(1) Exostosis of left auditory canal, and remarkably slight interference with hearing.

The patient was a gentleman aged about 45, a patient of Dr. Churchill, of Chesham. He noticed that after washing the left ear, water "remained behind," and caused him discomfort. On examination a sessile exostosis, the size of a small pea, was seen growing from the posterior wall of the canal, considerably diminishing the calibre of the tube. The hearing was little if at all impaired, and the patient suffered no marked inconvenience. No operation was, therefore, advised. So far as could be ascertained there was no cause for the growth in this case; the patient was not a bather or gouty, and he had never suffered from otorrhœa. There was no trace of growth in the opposite canal.

(2) Bilateral exostosis combined with old middle-ear catarrh.

A gentleman aged 42, from Johannesburg, had been getting deaf for many years. He heard a watch at only three inches on the right side, and the tuning-fork tests were very suggestive of loss of nerve power. The walls of the canal of the right ear were normal. The drum was quite opaque and retracted, and the light spot was absent. The left canal was almost entirely closed by multiple exostoses, but that portion of the membrane which could be seen was opaque and retracted. He could only hear the watch on contact on this side. He occasionally suffered from severe tinnitus.

In this case operation was not looked upon favourably on account of the co-existent mischief in the middle ear. Very slight improvement only could be promised.

(3) Extreme hyperostosis of the left auditory canal associated with vertigo and discomfort—difficulty of operation.

A medical man consulted me in February, 1896, for the following symptoms:—He was quite deaf in the left ear, with roaring noises and giddiness. These symptoms were of gradual onset, but had been much worse lately. He was gouty and of marked gouty parentage. On examination the entire bony canal was seen blocked with a dense growth of exceedingly hard bone, which had its origin posteriorly, and infringed anteriorly upon the front wall of the canal. In the right ear a similar condition was present, but in an early stage, and the hearing was good. An operation was performed on February 28th, under ether. The

auricle was turned forward by a semilunar flap. Pressure forceps were applied to the numerous vessels, and the cartilaginous canal was detached from the bone. The growth was now readily under observation and control. With a dental engine worked by my friend Mr. Barrett, I cut a deep groove superiorly into the growth at its junction with the bony canal posteriorly by means of a terminal cutting on the side. This being accomplished, the growth was seized with a pair of "stump" forceps and readily broke away, the whole proceeding occupying not more than fifteen minutes. Now, however, came the most difficult part of the operation. The portion of growth detached was only the outer part of what may well be termed a mound of ivory-like bone which extended right down the canal, having only a narrow "chink" between it and the anterior wall. It was obvious that this must be removed if any good was to result; and accordingly, with dental burrs, of which many were blunted in the process, I cautiously bored a canal inwards. The oozing was profuse and troublesome, and the operation most tedious and difficult. Towards the end, when a mere shell of bone remained, a guard was inserted beyond it, but the growth reached right down to the membrane, and practically a new canal had to be entirely formed. So soon as a free opening, the size of a quill, had been established, there escaped a quantity of inspissated secretion, which was removed by the syringe; some of this was of caseous condition, as though due to the discharge of an old otorrhœa. The operation lasted over an hour, and unless the auricle had been detached I doubt whether it would have been at all feasible. I was ably assisted by Dr. Clarence Blake in its performance.

The auricle was replaced and sutured with horsehair, and the canal filled with iodoform. The after symptoms were at first severe. The patient had intense pain and discomfort for several days, and the "noises" in the head only gradually lessened. Moreover, granulations sprang up from the newly cut bone, and it was not until April 17th that the parts were quite healed and dry, and a good inspection could be made. The new canal would admit a large crow-quill at its deeper part. There was a small perforation in the membrane, which was opaque and thickened. Hearing for conversation had improved, but he could only hear

the watch on contact. The sense of discomfort and the roaring noises had quite gone. His present condition is much as follows:—The hearing to voice and conversation is improved considerably, the general health is much better. There is still a small perforation superiorly; slight sounds, like the tick of a watch, cannot be distinguished, excepting close to the ear.

Remarks.—The essential difficulty in the treatment of this case, which was very great, was the extension of the bony growth inwards to the situation of the drum. From my experience of this operation I should think for long before I even attempted another of a similar nature, were I able to detect the nature of the case before operation. I have never before seen or read of a case exactly like it. The difficulty and risk of boring through solid ivory-like bone for a depth of some quarter of an inch from the surface of the bony canal is very considerable. The result, though on the whole satisfactory, is somewhat disappointing in detail. Although the more severe symptoms are relieved, the hearing is not restored in the satisfactory manner common in cases of exostosis. It is impossible to be sure whether the perforation in the drum was caused by the operation or not. Nothing occurred during its performance to make one believe that such an accident had happened, and the quantity of inspissated discharge that escaped seemed to render it probable that a perforation of ancient date existed, and that this was stirred into fresh activity by the operation.

The case affords a striking instance of the difficulties and dangers which may be unexpectedly encountered in the treatment of bony growths of the auditory canal.

The first two cases exemplify (1) that unilateral exostosis not associated with troublesome deafness scarcely needs interference; and (2) that old middle-ear catarrh may be associated with exostosis, rendering operation not very advisable.

The Unsuspected Nature of Serious Disease of the Middle Ear.

It is quite extraordinary, notwithstanding all that has been written regarding suppurative otorrhœa and its complications, how little the profession, as a whole, seems to appreciate the extreme danger and gravity of these cases. Among several that I have seen in the past six

months the following will illustrate how little patients regard purulent otorrhœa.

(1) Dangerous disease of the middle ear existing for long time untreated.

A gentleman, æt. 30, was sent to me by Mr. Buxton Shellito on July 4th, 1896. For a "long time," certainly for over ten years, he has had occasional pain and discharge from the left ear. He has never paid any attention to it, however, and only his friends complaining of the disgusting odour compelled him to seek advice. On examination the left canal was filled with offensive discharge, and in washing this away abundant granulation tissue growths as large as buck-shot could be seen springing from the tympanic walls. The drum was entirely destroyed. Bare bone could be felt on passing the probe. This was obviously a very dangerous ear. In such the complications of conchal abscess, meningitis, and lateral sinus pyæmia are especially apt to rise, from such causes as cold, bathing, or especially operations upon the tympanum.

(2) A young newly-married lady was brought to me by the husband on July 12th, on account of slight deafness. This was due to a slight catarrh. There was a distinctly offensive odour about one meatus, and on examination the drum was opaque and thickened, with two perforations separated by an intervening ridge of tissue. Considerable deafness existed in this ear. The patient now gave the history of "earache" as a child, with occasional discharge. She had previously thought nothing of the ear, and had actually engaged in the perilous practice of surf-bathing in her summer vacations.

Remarks.—Instances of this kind are common enough in practice, and there is still much difference of opinion as to how best to deal with them. A certain number of surgeons would in such cases extensively clear away the mastoid with strong burrs, and "curette" the tympanic cavity. The objection, in my mind, to such proceeding is (1) the uncertainty that a fragment of carious bone may not still be left about the inner walls or roof of the tympanum; (2) the extent and gravity of the operations; and (3) the fact that with care and attention to the ordinary modes of cleanliness such patients long survive, and may finally die of other maladies in no way connected with the ear. I believe the "expectant" plan of

treatment still holds its ground. Among the large number of local applications applied in these cases, none is, in my experience, so beneficial, when suppuration is profuse, as a ten-volume solution of peroxide of hydrogen.

Remarks concerning Adenoid Growths of the Naso-pharynx.

1. *Indications for Operation.*—Unfortunately, the greatest difference of opinion seems to exist as to the cases of this affection which necessitate operation. The presence or absence of attacks of deafness is an important consideration, and renders desirable the removal of even a small amount of lymphoid tissue from the region of the Eustachian orifices. Otherwise, slight impairment to nasal respiration, or the making of "noises" on the part of the child, as "sniffing," hardly justify operation, a simple nasal wash being in such cases all that is needful. Almost all delicate children who are brought up in large cities have an excess of lymphoid tissue in the naso-pharynx, and if this is a sufficient reason for operation, whole families may as well be subjected to the proceeding at once. Again, these growths seem to vary in size from congestion, and they may be prominent at some times and not at others.

2. *Method of Performance of Operation.*—In my own practice I never attempt to do this operation "against time." I always have full anaesthesia produced by means of gas and ether. The side position, with the head hanging over the edge of the table, is usually selected. The patient is allowed to respire for about a quarter of a minute, to lessen the congestion produced by the ether, and if there be any necessity a little chloroform vapour may be now blown into the naso-pharynx through a tube. In young children I always employ curettes of various shapes and sizes, followed by the steel nail, and finally my own nail. I take care as far as possible to scrape away every fragment of growth that is at all prominent. In older patients Lowenberg's forceps is usually employed, and as much growth as possible removed with it as a preliminary. Great care is needed in employing this instrument not to tear away strips of mucous membrane. I have seen this done on several occasions. The exact method of operating and position of patient differ in the practice of different operators. The mode of

operation is certainly not of such importance as the thoroughness and care with which it is executed. It is for this reason that I especially prefer the lateral position for these cases; be the bleeding ever so profuse, it flows away from the larynx into the dependent cheek, and the operation can proceed unchecked, to methodically clear away every fragment of growth.

After Treatment.—In the practice of most surgeons, as far as I can ascertain, no after treatment is advocated for adenoid growths. Considering that there is an extensive raw surface behind the palate, covered with inspissated blood and discharge, I have always aimed at keeping this clean and pure. So soon as the patient has recovered from the anaesthetic he is instructed to blow his nose to expel all clots, and in twelve hours the nares are gently syringed through into the throat with a warm solution of boric acid. The tubing at the end of the syringe should be carefully adjusted to the size of the nostril, so as not to fill it tightly, and thus force the fluid towards the middle ear. If there be any foetor about the breath on the second or third day, the naso-pharynx is gently sprayed with peroxide of hydrogen, or a dilute solution of Liq. Soda Chlorinatae. This is especially useful should the tonsils also be removed at the same time. In cases of deafness I always insist upon the regular use of the Politzer bag for a full month after the operation; and frequently order astringents to the throat, to be applied by sprays or brushes. The full benefits of the operation will not be obtained unless this is done.

Question of removing Adenoid Growths and Tonsils at the same Sitting.

As a general rule enlarged tonsils and adenoid growths can be removed at one and the same time. In weakly and anaemic children this rule may be relaxed, and the operations done at the interval of a month. The haemorrhage in these cases is sudden and profuse if the operations be thoroughly and not timidly or insufficiently done. Two guillotines should be at hand, and the anaesthetised patient brought into the sitting posture by means of pillows. The mouth being well opened by a Fergusson's gag, the tonsils are then removed as rapidly as possible before the bleeding from one obstructs the view of the other.

The patient is now turned into the lateral position, and the operation for adenoids proceeded with as usual. In these operations the aid of a skilled anaesthetist is an absolute essential. If, as commonly enough happens, the tonsils should be flat and ragged, I invariably remove them with a sharp spoon and the finger-nail, all forms of guillotine acting imperfectly in these cases. After healing, the galvano-cautery is applied to any hypertrophied masses of tissue that may remain. I have been much struck with the pains and care needful to get a good result in cases of flat and ragged tonsils.

Recurrence of Adenoids.—There can be no question that there is in some cases, usually children of a lymphatic type, a marked tendency for adenoid growths to recur. This is an additional reason for performing the operation very thoroughly. In several "recurrent" cases I have treated I find, from my notes, that the first operation was done without an anaesthetic, or under nitrous oxide gas, and hence was likely enough too brief in duration to be thorough. So far as I know, I have only had one case of recurrence in my own practice. A boy æt. 14, in whom adenoids had been very thoroughly removed two years before, got recurrence of symptoms; on examination with the finger there was distinct return of the growth, but not enough to justify a second operation.

Pulsating or "Clicking" Tinnitus.

An instance of this rare affection, the second I have seen, came under my notice in October, 1895. A young gentleman had suffered from a clicking noise in the right ear for some eight or nine years. He was slightly deaf on that side, but had no pain or discharge. On examination, beyond a little opacity of the membrane, there was nothing to be made out, but there was some loss of hearing power as compared to the other ear. On listening at the right meatus a ticking sound, exactly like a loud watch, could be distinctly heard. With the diagnostic tube it was most distinct and evident. The beats were synchronous with the pulse, yet occasionally they were intermittent, and by a powerful effort of will and moving the head and throat muscles he could check the noise altogether. It invariably recurred, however, and often came on when he could not help it, occasioning much distress.

A variety of treatment, including electricity to the palatal muscles, had been tried with no success. I elicited from him the interesting observation that he had been severely affected with stammering in early life, and this raised the supposition in one's mind that the clicking noise was due to some uncontrollable action of the palatal or tympanic muscles. In the *British Medical Journal* for November 16th, 1895, a very similar case to this was related by Sir William Dalby in a young lad æt. 15. He had met with two similar cases. A paper on this curious affection was read by Fitzgerald in 1881. All treatment of it seems to be very unsatisfactory.

ANTRAL SUPPURATION FOLLOWING INVASION BY A DENTAL CYST.

By J. G. TURNER, F.R.C.S.

THE following case serves to illustrate some of the points in the diagnosis and causation of suppuration in the antrum, and of dental cysts. A married woman, æt. 35, complained of great pain and swelling of the face of ten days' duration. There was an inflammatory swelling of the right side of the face, which included the upper lip, the cheek, side of the nose, and the lower eyelid. It was most intense and tender over the antral region. On examining the mouth, an evenly contoured bony swelling, yielding but not crackling on pressure, was found extending from the canine eminence to the region of the last molar tooth of the right upper maxilla. The alveolus, and with it the teeth, was depressed, and the remaining teeth were separated in a fan-like manner. There were no antagonising teeth in the lower jaw. There was no swelling of the palate. The bony swelling itself was only slightly tender, but the wall of the antrum above it, though not included in the swelling, was very decidedly so. The history of this swelling was that eight years ago the patient had had pain and swelling of the right upper jaw, which had subsided, but had recurred at intervals since. Eight months ago the present swelling was

first noticed in the region of the previous trouble; it had continued to increase up to the present time. At the onset of the present trouble two carious teeth, the pulps of which were dead, had been extracted in the hope of relieving the pain, but unsuccessfully, though pus was said to have escaped from their sockets. On inquiry it was found that there was "discharge of matter" from the right nostril, "worse in the morning," but the patient had no bad smell in the nose. There was no history of tubercle or syphilis, except that the patient's hair had fallen off in large quantities at the birth of her first child.

The nature of the bony growth had first to be decided. It might be solid,—probably then sarcomatous,—but seemed more likely cystic. The history was typical of dental cyst, and the yielding nature of the swelling pointed in the same direction. A soft sarcomatous growth, too, might have pulsated; here there was no pulsation, yet depression of the alveolus and irregularity of the teeth pointed to solid growth. It has been stated that there were no antagonising teeth in the lower jaw, and in estimating the value of this sign the difference must be appreciated between teeth which are "elongating" from disuse, being, indeed, cast off as foreign bodies, and teeth which are forced bodily downward together with the alveolus by force from above: in the one case the alveolus remains in place, and the tooth is gradually extended; in the other the tooth retains, as far as may be, its normal depth of implantation, and both are forced down together. This depression generally occurs with solid growths, and the strong alveolar border would be, at any rate, the last to yield to the pressure of a fluid swelling.

But, allowing the cystic nature of the present swelling, its origin outside the antrum would give it some considerable time to act on the alveolar border before pressure was relieved by opening into the antrum, and this may be supposed to have yielded for want of the support usually given by antagonising teeth. The swelling was a "dental cyst." Dental cysts are chronic cystic enlargements of the jaw, presenting occasional outbursts of acute symptoms, which arise in connection with "dead teeth," i. e. teeth which, following extensive caries, have suffered death of the pulp. The pulp putrefies, and the absorption of septic matter through the apical foramen of the fang sets up

acute inflammation which runs on to pus formation, hollowing out a cavity round the end of the fang ; the acute manifestations subside, leaving a chronic abscess of the bone in connection with the dead tooth : each access of septic matter through the apical foramen sets up another outburst of inflammation, varying in intensity according to the amount absorbed, and gradually a larger and larger cavity is hollowed out until it is evident as a cyst of the bone large enough in some cases to have thinned or even caused absorption of the bone over it, giving rise to sensations of yielding or crackling on pressure. Had a sinus formed at the onset and been kept open, no enlargement of the cavity could have occurred. Another theory of their origin postulates the same inflammation and consequent irritation of the tissues round, but gives quite a different starting-point. M. Malassey thinks they arise from remains of the enamel forming epithelial involution, which, under the name of paridental epithelial remains, he describes as occurring as small masses of epithelial cells scattered normally through the alveolo-dental ligament uniting the tooth to its socket. These, he says, proliferate when irritated, as by septic inflammation in their neighbourhood, and form masses and tubular processes, the masses often becoming hollow in the centre. Hence he is able to explain the origin of these cysts as due to the irritation of the epithelial remains by the septic inflammation, and to explain their occasional occurrence at some distance from the tooth with which they seem to be connected by assuming cystic formation at the end of one of the tubular processes of epithelium. This theory requires the cavity to be lined with epithelium.

In the case under consideration a diagnosis of dental cyst was made, and it was surmised that it had invaded the antrum and in some way set up suppuration.

In considering the state of the antrum it had to be noted that although there was free discharge through the nose, yet there were great pain, tenderness, and swelling over its walls,—symptoms not usually met with in ordinary chronic antral suppuration unless the ostium maxillare becomes blocked. The explanation seemed to be that the bony walls of the antrum were diseased, probably necrosing, and that the bone disease had set up its symptoms of pain, tenderness, and swelling,

and was the source of the greater part of the trouble.

In a case of suspected antral suppuration, the proof of pus in the antrum is often very difficult. In a case where the ostium is patent there may be some slight swelling and tenderness of the face over the antrum, and a deep-seated uneasy feeling ; there may be actual pain, often referred to the frontal region, though this is more likely to occur when the ostium is blocked ; there may be a history of discharge from the nose, sometimes worse in the morning, because the patient may have slept in such a position as to retain the fluid in the antrum during the night, moving into a position favorable to its escape on waking ; on the other hand, the pus may have trickled down the throat, giving rise to a foul taste in the morning, and to dyspepsia. There may be some inflammatory swelling of the mucous membrane of the turbinate bones due to irritation of pus passing over them, and a smell in the nose noticed only by the patient. Further investigation may be made by asking the patient to assume a position in which the head is somewhat dependent, and the suspected antrum uppermost ; the patient may be asked to sit for a short time with the body well bent forward and the head turned on the side, and then to blow his nose ; also by transillumination and by exploratory puncture.

In transilluminating an antrum the room is well darkened, or a photographer's cloth is thrown over both heads, a small electric light is put in the patient's mouth, and the lips shut. If the antrum is normal, light shows through the orbital plate of the maxilla, through a small portion of the front wall of the antrum below the orbital margin, and to a less extent through the whole side of the face. The nose and parts just beside it are well lit up. The important place to look for light is at the outer part of the orbital plate. Both sides should be compared. If there be fluid or growth in the antrum, or if the bony walls or soft parts be thickened, as by inflammatory effusion, less light will pass through. As applied to suppuration of the antrum its chief value is as a negative sign, since even inflammatory effusion stops the transmission of light. In exploring, the nostril should be cocained, and a small trocar and cannula thrust obliquely through the outer wall of the inferior meatus from the front of the nose. A suction syringe fitting the cannula should be at hand, as the

fluid may be too thick to flow through a small cannula. If pus is drawn it is conclusive, but it must be remembered that pus may be present at some times and not at others.

In cases in which the ostium is blocked there can, of course, be no symptoms caused by discharge flowing from the nose. The course of events is more acute; pus soon becomes confined under pressure and causes great pain, tenderness, and swelling, as well as constitutional disturbances. These cases form a minority of all antrum suppuration cases.

An adequate cause should be inquired for. In strumous subjects a blow on the face may be sufficient, but usually the teeth will be found to furnish the cause. A tooth to set up antral trouble must be dead, *i. e.* its pulp must have died, so that septic absorption through its apex may be possible. I have never seen a paridental abscess—which may occur along the side of the fang of a living tooth—lead to antral trouble, the pus finds a ready exit round the neck of the tooth. The most likely teeth are the bicuspids and molars, the fangs of which are in close relation with the floor of the antrum, and in some cases even project into its cavity. In the latter case, if septic matter passes up through the apex, antral suppuration may be at once set up, and be the first indication of dental trouble. If there is some thickness of tissue between the tooth and the antrum, a virulent septic inflammation may easily destroy the bone and cause perforation into the antrum. The antrum, as in the case detailed, may be invaded by a dental cyst, and here again the tooth must be dead to form the starting-point of such a cyst.

In the case before us, on removing the outer wall of the cyst the floor of the antrum was found to have been absorbed, and the inner wall of the antrum to have almost wholly disappeared, so that a finger introduced through the cyst readily met one introduced through the nostril. No bare bone was found; but a few weeks later the patient complained of a bad smell in the nose, which was not noticeable to those around, and mucous polypi were found hanging down from the antrum into the remains of the cyst cavity, and rendering the antrum dark on transillumination. They were removed, as well as some small pieces of dead bone from the antrum, and its outer and back walls were found rough and bare. It was surprising

to note how quickly these polypi developed. In a period of three weeks, at the beginning of which there was no sign of them, they had filled the antrum and were hanging into the cyst cavity. They were an evidence of the chronic irritation of the purulent discharge.

The pain and tenderness are now gone, but the swelling remains to a considerable extent, and probably will not subside till more bone has been cast off. In the meanwhile the patient has been taught to syringe for herself, and to pack the opening into the cyst cavity with iodoform gauze to keep it patent. She is also taking iodide of potassium. The cyst wall was examined and found to be lined with epithelium, showing the importance of thoroughly removing it.

TREATMENT OF APPARENT DEATH IN THE NEWLY BORN.

BY PROFESSOR PINARD,

Professor of Obstetrics, Faculty of Paris; Accoucheur to Baudelocque Maternity, etc.

GENTLEMEN,—The woman you saw confined yesterday presented certain dangers that we will do well to avoid if possible, and we propose to go over her case with you, as it is an instructive one. She was confined for the first time in July, 1893, in Professor Tarnier's clinic. It is probable that there was some intervention then, but she does not seem to be aware of or remember what it was. On examination we found that the promonto-subpubic diameter was equal to 10·3; the head was high up. We supposed that the placenta was inserted on the inferior segment of the uterus.

Added to this a large quantity of amniotic liquid, and you have a trio of unfavourable prognosis in the case,—that is placenta prævia, hydramnios, and vitiated pelvis. The first pains came on two days ago; the labour went on quietly, and the os uteri dilated slowly also, while haemorrhage occurred from time to time. The pains increased, and the head still remained high up, while the bag of waters came down and out.

Fearing a new haemorrhage we proceeded to

rupture the membranes, and hoped that the head would descend. A large quantity of liquid came away, but the head did not come down, and we tried to discover the presentation. Thinking we felt the eye, we made a diagnosis of face presentation, and then fearing that Champetier's bag, or any other intervention, might displace the head and make our difficulties worse, we decided to wait.

The result was, that during the day the contractions became violent, the head descended flexed, and a child of seven pounds weight was born.

The mother's temperature, which had been up, fell to normal, and she is now quite well. So that is a great success to report for conservative midwifery. For the woman was in greatest danger from haemorrhage, which might have caused her death.

But the infant came away in a state of apparent death. Now, without stopping to define what we mean by this expression, we may say that there are several different causes. First, the child may be born with a respiration that is not regular, and the heart's action accelerated, and in a little time this will arrange itself, the child having been born, astonished, as it were, by the force of the muscular action of ejection.

In other cases the infant may be born with the heart beating, but no respiration; or, again, there may be neither heart-beat nor respiration. But do not say that it is dead; you do not know. The death may only be apparent, not real.

As to the present child, its heart beat, but there was no respiration. We adopted several methods to revive the baby, but without success. Let us profit by this case to detail the proper method of acting in similar ones.

At this Baudelocque Clinic we had, from December, 1892, to 1894—that is to say, in two years—fifty infants born in a state of apparent death. Twenty-four of them were brought to active life, and were saved by clearing the obstruction of the respiration and by stimulation.

You know already the simple methods we use here. As soon as the child is born we introduce a finger into its mouth to get out the mucus that is sometimes in the way of its breathing properly. As to stimulation it is very important in these cases, and we recommend a useful method of

rubbing the vertebral column. This friction of the spine is one of our best treatments. You will often see with it a child in apparent death open its eyes and at once begin to breathe. It should be done with a certain amount of vigour. It is well to spank these babies, but it must be done with care, for we find it often causes ecchymosis; and be careful also in wrapping them in very hot cloths, as we have found some of them badly burnt by this system. If in spite of all these methods the child still does not breathe, it is necessary to commence insufflation. It was Chaussier who first thought of a tube to blow into the larynx some air, and Ribemont modified his instrument into the present one we use. He was able to give it the proper anatomical proportions and its curve.

Ribemont's tube is then introduced into the mouth, and from there to the larynx, guided by the finger; then the mucus is drawn up and out, and you proceed to blow the air into the lungs. There are some difficulties and a danger in using this tube, but do not suppose that we advise using the mouth to mouth method; this is not at all good. It is thought that air can be passed into the lungs in this way, and yet it mostly passes into the stomach; indeed, we once saw a case where the air went on and came out at the anus.

The danger of insufflation consists in the production of emphysema owing to the rupture of the pulmonary vesicles. This has been disputed, but we have been able to prove it, and, indeed, it is natural when you think of the fact that you have to do with lungs that have never as yet breathed, and are in a state of atelectasia. When you blow air into the lungs, it does not penetrate equally or in a uniform manner, so that some of the vesicles are apt to rupture as they receive the greater pressure of air. It is for this reason that Ribemont advises us to make the insufflation slowly, without any effort. If you do this, you have in your hands the best method of reanimating such infants. It was this method that we used in seventeen of the children, and though eleven of them died afterwards, respiration was re-established in all of them.

In six others we tried Laborde's method of rhythmic traction of the tongue, but all of them died.

In certain cases where the heart was still beating Laborde's method brought about the recoloration

of the skin ; but the respiration was not re-established, and the heart failed. We then took up insufflation, and were able to save some of the children. Must we conclude from this that M. Laborde's method is useless? Not at all. If we found ourselves near a drowned man we should use it. It, indeed, is good in adults, but in the special cases of lungs that have never yet breathed it does not seem to be destined to be of much use. You might try it when all else has failed, but we could not get any success with it in infants born without respiration in a state of apparent death, so we cannot recommend the method. In any case do not use instruments ; the fingers are the best in such cases when you can apply the method to adults.

To sum up the result of our experience, we have found that the best method in apparent death in infants newly born is the use of Ribemont's tube to perform insufflation of the lungs. We have, indeed, seen the simple contact of this tube make the reflexes act, both of circulation and respiration, and in using this system you will save the lives of many such infants ; but never forget that it is an instrument that has a certain danger, as we have shown, and be careful to use it slowly and with prudence.—*Medical Fortnightly.*

THERAPEUTICAL NOTES, &c.

Infectious Icterus in Children.—Ulrik relates three cases of contagious icterus in children, 7, 8, and 10 years of age, all of whom presented the same symptoms—anorexia, fever, and icterus,—which set in after the decline of the fever, and which continued only for a few days. Two of the children belonged to the same class in school, and the third came daily in contact with the first child ; none of the grown-up members of the families were taken ill. Small epidemics of contagious icterus are not rare in Denmark. Ordinarily the disease is not severe, but it may be so, and has caused sudden death.—*Ugeskrift for Læger.*

Rachitis and its Relation to Dampness.—Hagen-Torn, in a study of this subject, comes to the following conclusions :—1. Rachitis is an endemic disease, depending on the degree of the yearly dampness in the respective country. By this is also explained the oscillation of its frequency in connection with the season (increase in winter).

2. Disorders of digestion are most frequently the consequence of the disease. 3. Rachitis is accompanied by changes in all the organs. 4. Disorders in nutrition of the organism and in the development of bones are the consequences of disorders of general metabolism. 5. The disorders of metabolism, however, are occasioned by the retention of the dampness in the tissues of the body. 6. In localities where the yearly average dampness is higher than 80° F., rachitis is, as the author expresses it, a physiological condition. In localities between 80° and 70° F. of dampness it develops only in specially unfavourable conditions of lodging and care of children. In localities, however, with dampness below 70° F. rachitis does not occur.—*Vratch.*

Hæmorrhage in Brain Surgery.—The control of hæmorrhage is one of the most difficult problems in connection with the removal of cerebral tumours. Hæmorrhage from the diploë is easily controlled by Horsley's antiseptic wax. For hæmorrhage from the vessels of the meninges the ligature is an efficient means of control. If the dura be cut and an artery bleeds, the cut end can be tied just as any other vessel. If it be necessary to ligate a vessel in its continuity, the dura being unopened, though with torn vessels, it can be secured by passing fine silk thread by means of the finest semicircular Hagedorn needle under the dura and around the vessel, care being taken not to wound the underlying cerebral veins themselves. For venous hæmorrhage, the best method also is the ligature. Rarely can the vessel be seized by the forceps and a ligature applied. Pass by means of the semicircular needle of suitable size a silk or catgut ligature through the cerebral tissue immediately below and around the vein, and then tie the vessel by drawing with equal force the two ends, not constricting the vessel with so much force in tying the knot as to tear through its weak walls.—KEEN, *International Medical Magazine.*

The Treatment of Acute Croupous Pneumonia.—In the *Revue Internationale de Médecine et de Chirurgie*, CASSINE contributes a paper with this title. He points out the absolute necessity of placing the patient in a large airy room ; the sputum should be received in cloths, which should be immediately destroyed, and the condition of

the mouth, nose, and intestinal canal should be carefully looked after during the entire course of the illness. Mild diuretic waters are of value for the purpose of maintaining urinary secretion, and of washing toxic materials from the body. Broths are also useful in the way of nourishment if they are properly made.

When the disease is once thoroughly established it is often necessary to support the heart, which is apt to flag under the combined influence of the obstruction to the flow of blood in the lung, and the toxic materials produced by the micro-organisms. To overcome this cardiac failure, digitalis is useful either in the form of the infusion, the fluid extract, or the tincture, the dose of the last preparation varying from five to twenty drops. Should the heart show evidences of failure, it is well to employ subcutaneous injections of a solution made as follows :

B. Boiled distilled water, 3 drachms ;
Caffeine,
Benzoate of sodium, of each 40 grains.

Two or three hypodermic syringefuls of this solution may be given in twenty-four hours. For the reduction of the temperature numerous methods may be resorted to. Antipyrin is not to be advised, because of its influence upon the blood. Sulphate of quinine may be used as an antipyretic, and for supporting the system, in the dose of two to three grains; but very often as much as thirty to forty grains is necessary before any antipyretic influence is felt, and such doses often depress the heart. Cold compresses may often be applied to the chest with advantage, but cold baths can only be employed in a limited number of cases.

Should the pneumonia attack a person who is markedly feeble by reason of old age, excessive use of alcohol, or similar causes, it is of vital importance that the nervous system and the heart be supported. Under these circumstances Cassine believes that digitalis is by no means as useful a remedy as caffeine, and that with caffeine we should administer alcohol, the acetate of ammonium, and ether, and even perhaps such drugs as kola, coca, and nux vomica. Counter-irritation in the form of blisters is not to be applied in this class of cases.

Should croupous pneumonia attack a child, care should be taken to maintain the strength by the

proper administration of nourishment, by the maintenance of free diuresis, and by careful attention to the condition of the alimentary canal. Small doses of alcohol, or even of digitalis, may be used if they are necessary; and if the temperature is excessive, and is associated with shortness of breath, insomnia, and cerebral excitement, a hot bath repeated four times a day, to which has been added a small quantity of mustard, will prove of value.

In the great majority of cases of croupous pneumonia occurring in children the illness is not exceedingly severe, and recovery takes place.

Therapeutic Gazette.

The Diagnosis of Tuberculosis from the Morphology of the Blood.—A. M. Holmes, A.M., M.D., in a contribution on the above subject, concludes as follows :—“First, as the tuberculous condition becomes more marked and the gravity of the case increases, the percentage of small lymphocytes decreases, and the percentage of phagocytes increases.

“Second, as the tuberculous condition becomes less marked and the convalescence increases, the percentage of small lymphocytes increases, and the percentage of phagocytes decreases.

“Many other deductions might be made from the tables with reference to the physical condition of the patients. For example, in all cases in which the blood shows the usual tuberculous characteristics, together with an increase in the phagocytes to eighty per cent. or over, it is quite safe to diagnose advanced pulmonary tuberculosis with cavity, profuse expectoration, and abundant bacilli; or, if surgical tuberculosis, an abscess with more or less discharge of pus.

“I do not wish to be understood as saying that all of the appearances that I have described are necessarily present in each case of tuberculosis. Neither do I wish to say that many of them are not found in other diseases. But I do claim that a peculiar combination of blood appearances is characteristic of this disease, to the extent that they will enable us to make a diagnosis at an earlier date than by any other means that we now possess. It is quite probable that the near future will justify a stronger statement: that from the blood condition we will not only be able to diagnose tuberculosis, but that in many cases it will enable us to distinguish the various stages of

the disease, and hence will be a valuable means of following the course of the disease under various methods of treatment.

" In conclusion, I will review this study with the following summary:—That the diagnosis of tuberculosis, from the morphological appearance of the blood, rests upon the hypothesis that each individual has a biological prototype in the leucocytes of his own blood. That leucocytes are independent organisms with functions analogous to those of the larger organism. That they pass through stages of growth and decay. That disintegration of leucocytes may occur at any age. That the leucocytes are tissue formers. That as are the leucocytes, so is the individual. That tuberculosis is a disease characterised by tissue disintegration. That in tuberculous blood there is abundant cell disintegration, premature development, premature decay, and more or less deviation from the normal percentages of the various types of cells. That if there is marked disintegration in the leucocytes, it is with absolute certainty that we can predict a similar condition in the larger organism. That tuberculosis possesses a combination of blood appearances, from which a diagnosis may be made earlier than by any other means that we now possess. That these may be recognised by appropriate micro-chemical stains and under a high power. That they can be recognised even before the disease manifests itself in the individual. That they are sufficiently marked in tuberculous persons, or even in those with a strong tuberculous predisposition, to enable a diagnosis being made from the blood alone, without knowledge of the history or physical condition. That the real source of the recuperative power is to be found in the leucocytes. That thus far no other pathological condition has been found which presents similar blood appearances. That to secure an early diagnosis would enable many to avail themselves of favourable climatic changes, and thereby delay or even prevent the destructive results which would otherwise inevitably follow. And, finally, that if future investigations confirm these deductions, we may look forward to a no distant day when, if we expect to detect tuberculosis in its incipiency, we must study the leucocytes."

Medical Record.

Modern Treatment of Eclampsia.—Dr. T. Marx, one of the surgeons to the New York

Maternity Hospitals, advocates measures which are both timely and radical. His indications for interference he bases not upon the presence of albumen, oedema, or constitutional symptoms, but upon the amount of urea excreted in twenty-four hours. The ætiological factors he discusses somewhat at length, but comes to the conclusion that the pathogenesis of eclampsia is surrounded by a mist of doubt. He divides the toxæmia of pregnancy into (1) That complicating heart disease. (2) Acute pregnancy nephritis. (3) Acute exacerbation of a chronic nephritis. (4) Acute toxæmia in which neither albumen nor casts appear, which he terms "urinæmia."

In cases under heading (1) he advises constant administration of heart tonics, and where these fail, or in any case where albumen and casts appear, induction of premature labour, preferably in one sinking under chloroform, the cardiac affection being no contra-indication to giving chloroform.

In cases under heading (2) we have a full and complete therapeutic measure—one not nihilistic; rest, milk, hot sweating, heart tonics in full doses (those that irritate kidneys, like digitalis, iron, etc., contra-indicated), caffeine, nitro-glycerine, sparteine, are among those advocated.

Pilocarpine he mentions only to condemn. He advises early operative interference, and, as stated above, he places his indication when, in spite of vigorous scientific treatment, the amount of urea excretion remains stationary or progressively diminishes.

After labour large doses of morphine, where the kidneys are active, to control convulsions, further asserted by the inhalation of chloroform.

Under heading (3). In this large class of cases he admits of brilliant results in the continuous administration of from $\frac{1}{100}$ to $\frac{1}{50}$ gr. of nitro-glycerine from the first months of pregnancy. He has succeeded in carrying a number of women to term without any bad symptoms.

With this treatment he claims it is not necessary for a mild diet, since he advises a liberal diet throughout pregnancy.

In class (4), cases called *urinæmia*, the diagnosis is of great importance. The diagnosis can only be made when symptoms pointing to a toxæmia be present and a diminished urea excretion is found. The only treatment advised is prompt emptying of uterus, and then treatment as above.

The Charlotte Medical Journal.

THE CLINICAL JOURNAL.

WEDNESDAY, OCTOBER 21, 1896.

CLINICAL DEMONSTRATION ON A CASE SUITABLE FOR THE OPERATION OF RADICAL CURE OF HERNIA.

BY
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Assistant Surgeon to St. Bartholomew's Hospital, Surgeon
to the Great Northern Hospital; &c.

WHEN I demonstrated the case, which was unsuited for the Radical Cure of Hernia,* it was said that we should soon meet with one which was suitable.

You may remember that great stress was laid upon the proper selection of cases for the operation. Done upon the right kind it is a brilliant success, highly appreciated by the patient, and affording much credit to the surgeon ; but done upon wrong ones it ends in relapse, brings everyone into discredit, and does much harm to surgery in general. However, you have seen an improper case, now let us look at a proper one. It happens that the patient before you was operated upon three months ago, and has now come up to report himself, and see if he is ready for hard work. This, however, does not make him less suitable for our purpose. We will begin as usual by looking at the *whole* patient. He is a young man with all the appearances of robust health. His complexion is clear, his cheeks and lips red and wholesome, his eyes bright, and his hair glossy. He stands erect, and moves with rapidity and decision. His step is firm and springy. Any one at a glance would say that nothing ails him, and that all his organs are sound,—a fact of which we definitely assured ourselves before the operation. Now examine the abdomen. He has a straight clean-cut scar over the right inguinal canal. That is where three months ago he had radical cure done for an inguinal hernia. He now comes in accordance with the usual instructions to report himself at the

end of three months before beginning hard work or severe exertion. His hernia is cured, but when it was present its characters had very little bearing upon the question of operation. Had the inguinal canal been enormously distended and obliterated, the operation would have been harder, but that would have been all. Now turn to the rest of the abdomen. The skin is supple, clean, and white, and betokens healthy tissues beneath. When you pinch the skin up it feels elastic, and very little subcutaneous fat is felt. Young men ought not to be laden with fat, and when they are their tissues are not as good as they ought to be. I do not decline to operate upon a young man because he is fat, but they are much more likely to be troublesome when under anaesthetics and after the operation, and their tissues are not of really good quality. If now we study the shape of the abdomen, you will observe that it is flat—that is to say, there is not, as in the other case, any protrusion of the lower part, or sinking in of the upper. Nor can we find those bulgings at each side of the rectus which indicate that the hypogastric fossae have become like funnels leading to the inguinal canals. The semilunar lines at the outer sides of the recti are clearly defined, and all his muscles are well developed. For instance, the costal origins of the serratus magnus stand out quite clear and distinct. This young man's abdomen is like that of the statue of Antinous, with which you are all familiar ; but which, by the by, has always seemed to me to be the presentiment of one who was rather too fat and well fed.

You may remember that the history of the unsuitable case threw light upon the pathology of his rupture. Histories are to be taken with great caution and incredulity. Often they are mere echoes of the "leading questions" (as lawyers call them) which have been asked by the credulous. But in this man's history one fact was volunteered which deserves great consideration. Here are the notes of his case, and they say that seven years ago, when he was twenty years old, his attention was first called to his rupture by an attack of acute intestinal obstruction and the

* "Clinical Journal," June 24, 1896.

simultaneous appearance of a lump in the right groin. A medical man fortunately succeeded in reducing this strangulated rupture, for such the lump was, and afterwards he was confined to bed for a week. Now this is quite a common history in cases of congenital hernia. The man has no defect whatever of his tissues, or of the suspensory apparatus of his intestines and viscera, nor has he any elongation of the mesentery as used to be supposed. His hernia was due to a developmental defect in his abdominal wall. Under ordinary circumstances the peritoneal canal, down the back of which the testicle travels into the scrotum, closes up after the gland has reached its destination. But, not infrequently, the peritoneal canal, or processus vaginalis, never closes. Often the first intimation of this failure is the sudden appearance of a hernia. It is very strange that the processus vaginalis may remain unclosed for years, and, for some odd reason, nothing pass into it from the abdomen, although the mesentery is always long enough to allow this occurrence, and although the great omentum, too, is often sufficiently long. Until, at length, a day comes, as in this case, when a knuckle of intestine suddenly slips into the patent processus vaginalis. Inasmuch as the canal is narrow, with unyielding walls, the most acute strangulation is apt to supervene, and unless prompt and skilful aid is at hand the unfortunate patient gets gangrene of the bowel and probably dies. It is unnecessary to point out how this is borne out by the history of the case of the man before you.

Warned by the danger he had escaped, he wore a truss. This instrument seems to have been less irksome to him than is usual; but his rupture appeared again to some extent when his truss was taken off at night. If you listen to some you might suppose that trusses were divine instruments, which people wore because of the pleasure they afforded. How such erroneous views can be acquired or propagated I cannot imagine. Not only is a truss most expensive and troublesome, but it is also a grave social disadvantage. What man or woman does not conceal the fact that he or she wears a truss? Who is there who does not own with reluctance that he wears one? Moreover, when I went over the cases of strangulated hernia which had been treated in St. Bartholomew's Hospital during

about fourteen years, I found it said over and over again that the patient was wearing a truss which broke whilst lifting, and so the rupture came down and was strangulated; or, that the truss failed to keep the rupture up, with the same result. Next time you are told that a truss is a charming instrument, think how you would like to wear one yourself.

However, this man's hernia happened to be incomplete and easy to manage, and so he was contented, perhaps, to endure the dangers and disadvantages of a truss and all its false security, until, at last, he wished to enter government employment. It is somewhat significant that the government services will not accept the wearers of trusses, but insist upon the hernia being cured by operation. When this man came I had not the slightest hesitation in promising to cure his rupture, so that he should be accepted by the examining board. Usually I like two months to elapse betwixt the operation and the medical examination; but two years ago I operated upon a surgeon who passed into the army six weeks after the operation.

I see it has been noted that at the operation the hernia of this patient was *probably* congenital. The slight uncertainty in the surgical registrar's mind arose from the fact that the sac was of the funicular variety. When the hernia is of the ordinary congenital kind a finger can be pushed down the sac after it has been cut across, and the testis felt projecting into the back of the part which is normally shut off to form the tunica vaginalis.

If now you ask this patient to cough, no impulse can be seen where the hernia used to be, nor can any localised impulse be felt. I say *localised* advisedly, because I have seen the general impulse which a cough imparts to the abdominal wall mistaken for the localised impulse of a hernial protrusion. Moreover, he has no pain whatever; the tip of the finger will just enter his external abdominal ring, his scar is white and hardly to be seen, and he is sure to be accepted.

The operation by which this result was obtained is founded upon rational and physiological principles. Each step is guided by vision. The inguinal canal was laid open from end to end by a cut through the external oblique aponeurosis and the structures over it. I used to be reluctant

to divide the external oblique, lest it might fail to unite. But I gradually found this fear to be unfounded; indeed, the fibres of the external oblique aponeurosis are arranged so as to close together when put upon the stretch. Moreover, I have a lively recollection of a youth upon whom I operated without dividing the external oblique, and who afterwards had a relapse of his hernia. At the second operation, which I did at his earnest request, it was clear that the upper inch of the hernial sac had not been obliterated by the ligature which had been applied without the aid of vision.

We need not now discuss the operation at length. You have very many opportunities of seeing it done. Briefly, the canal having been opened, the sac is found and opened. Next the contents of the sac are dealt with—intestine reduced, and omentum transfixated, tied, cut off, and the stump returned into the abdomen. Next the sac is cut across and separated from the cord as far as its junction with the peritoneum, its stump transfixated and tied, and then fastened beneath the muscular fibres of the internal oblique and transversalis. This accomplished, the back of the inguinal canal is repaired with two or three stitches put behind the spermatic cord, and binding Poupart's ligament to the conjoined tendon and arching fibres of the internal oblique and transversalis. The operation is completed by closing the canal and the wound in the skin. The lower end of the sac is usually left to shift for itself, and I have never seen any ill results from this procedure. Sometimes the lower end of the sac is removed if it is small and unadherent to the spermatic cord. Sterilised silk is used for all the sutures and ligatures, except those in the skin, which are of silkworm gut, or fishing gut, as it is often called.

The after progress of these cases is very uneventful. For the first twelve hours they are upset by the anaesthetic and exposure, but have no pain in the wound. Usually they complain of pain in the back and beneath the costal cartilages. These pains soon depart, and after the bowels have been moved any light diet, with tobacco and wine or beer in moderation, may be given. The dressing is removed about the tenth day. We then teach them what Mr. Stanmore Bishop has called my rule, which is "three weeks in bed, three weeks on a couch, and six weeks'

gentle exercise." During the second three weeks the patient walks a little, and goes for a drive in a carriage. During the last six weeks he can follow a light occupation, but avoids the violent exercises such as riding, football, and so forth.

The danger of the operation of radical cure has been much reduced. It is now probably less than one per cent. I have never yet lost a patient from anything connected with the wound, but I grieve to say I have had a death from chloroform, another from ether bronchitis, and another from syncope a few hours after operation. My only consolation is that all the fatalities were in patients with irreducible herniae. The last sixty or seventy cases have been absolutely uneventful, and have not given us a moment's anxiety.

This patient naturally inquires whether he will require a truss. The answer is "no." He also wishes to know whether he will ever have another rupture. All you can say is that he is now cured and like other people, but that if he exerts himself beyond his strength, lifts enormous weights, and so forth, he may, like anyone else, acquire a fresh rupture. My own experience, which is now very considerable, is that efficient operations done upon suitable patients are not followed by relapse, and that the patient is delighted with the result, and is exceedingly grateful.

A POST-GRADUATE LECTURE
ON
CHRONIC GLANDULAR DISEASE
OF THE
NOSE AND NASO-PHARYNX.

Delivered at the London Throat and Ear Hospital, Great Portland Street,

By GEORGE STOKER, M.R.C.P.I., &c.
Surgeon to the Hospital.

GENTLEMEN.—The subject which we have to consider this evening is Chronic Glandular Disease of the Nose and Naso-pharynx, and I propose to allude, first of all, to what is known as hypertrophy of the tubercle of the septum; secondly, to post-nasal growths or naso-pharyngeal adenoma; thirdly, to enlarged tonsils; and fourthly, to follicular pharyngitis.

It is convenient to discuss these subjects to-

gether, firstly because they generally occur together; secondly, because if they do not actually occur together, the symptoms of one condition, arising singly, gives rise to many symptoms which are represented by another condition arising singly; and thirdly, it is convenient because the treatment for one very often involves the treatment for other of the different conditions.

On the septal wall, about the centre of the middle turbinated bone, there is a mass of glandular tissue, which is known as the tubercle of the septum. In cases of chronic catarrh from any cause, this tissue becomes hypertrophied, and gives rise to a train of symptoms which, on examination, you would not be able to account for if you did not recognise this hypertrophy.

There is difficulty in nasal respiration, sneezing, and evidences of chronic catarrh. On examining the nose, you would see a comparatively large mass of glandular tissue, which at first sight might be mistaken for an angle or deviation of the septum; but if you gently press it with a probe, you find it is a soft, yielding structure, characteristic of such glandular swellings. The only treatment for this form of trouble is the application of the galvano-cautery, in applying which it is advisable, for the comfort of the patient, to cocaineise the part. Here I would like to warn you very seriously against using the cocaine spray in a careless manner, as must be the case in spraying the nose, because a great deal more cocaine is used than is necessary, and dangerous symptoms arise through the patient swallowing some of it. The proper method is to steep a small peldorf of wool in a 20 per cent. solution of cocaine, pass it into the nostril, leave it against the part five or six minutes, then withdraw it, and with a probe touch the part forcibly and ask if the patient feels it. If not, he is not likely to feel you using the galvano-cautery. When withdrawing the cautery after the operation, you must be very careful not to touch the orifice of the nostrils with it. Even the wire, though not very hot, will cause much pain if allowed to touch the walls of the aperture. A further point is that the cautery must be *gently* disengaged from the part.

Next we will consider naso-pharyngeal growths. There is a large quantity of glandular tissue in this region, and hypertrophy of it very often, from one cause or another, causes post-nasal growths.

Among such causes is heredity; there is certainly a disposition, in a great number of children, probably owing to family history in part, as well as to the circumstances in which they live, so that what is an influence to one is common to all. Then there is the incidence of the exanthemata, which often set up chronic catarrh in the nose. The symptoms which post-nasal growths give rise to are very prominent and easily detected. Unfortunately over 50 per cent. of my cases come, not because they have any trouble in the nose, they generally seek advice for ear trouble. In most cases suppurative middle ear disease occurring in young children is to be traced to the existence of post-nasal growths. Therefore in every case of chronic middle ear disease occurring especially before adult life it is your duty to examine for post-nasal growths. The appearance of the patient will serve to put you on the alert—the pale, flabby face, open mouth, everted lips, broad, flattened bridge of the nose. In some cases this appearance is so marked as to be recognisable at some distance. On questioning the parents it transpires that the child is a mouth-breather, both in the day and night, giving rise to snoring while asleep. The resulting symptoms sometimes affect other parts—there is the nasal voice, attacks of some form of bronchitis with chronic cough; there is often indigestion in consequence of this constant secretion of thick mucus at the back of the nose being swallowed by little children; anaemia is also often present, owing to lack of proper oxygenation of the blood. Having got the history you will next proceed to examine. In a child it is always necessary to observe the condition of the pharynx and naso-pharynx as far as you can see. The pharynx will be found to be dry, and on depressing the tongue and requesting the child to say “ah,” you will find, in marked cases, paresis of the soft palate. This arises either from the fact that the growths are situated low down and prevent the soft palate going upwards and backwards, or the paresis is due to the physiological idleness of the soft palate. The examination can be made by means of a mirror or by digital examination. In using the post-rhinoscopic mirror, especially in children, do not examine the pharynx first, because this will make the parts irritable and prevent an inspection of the post-nasal cavity. Take care not to pass the spatula further back than

the anterior half of the tongue. My own experience is, that in young children there is only one way to ascertain the existence of post-nasal growths, and that is by digital examination, and I would like to mention some points in connection with this at the risk of some gentlemen present knowing them already. I have seen the finger passed into the mouth on many occasions, and turned up before the posterior wall of the pharynx is reached, so that the nail impinges on the soft palate, and of course no growths are felt. You must be careful not to bend the point of the finger upwards till you have reached the post-pharyngeal wall. Having discovered the existence of such growths, what treatment should be adopted? In my experience there is only one course which will effectually cure the patient, and that is the removal of the growths. Before the operation I think it is desirable to use a lotion to wash the nose and naso-pharynx twice a day for a week or ten days. When the lotion is syringed in, the patient should be told not to syringe towards the eye, but after inserting the point, to direct the barrel horizontally and towards the ear.

Next, I will say a word or two on the subject of anæsthetics. Some people are accustomed to operate simply after painting the pharynx and naso-pharynx with cocaine. To remove post-nasal growths from a child without an anæsthetic, in my opinion, borders on brutality. I do not think one can produce sufficient local anæsthesia with cocaine; and even if that were possible, the probability is that the growths would not be removed at one sitting. If the child becomes terrified at the first sitting, this militates against success on a subsequent attempt. Therefore I refuse to operate in these cases without a general anæsthetic. That which I use in children under four years is A.C.E. mixture, and over that age I prefer either that or gas, followed by ether. A most important point in regard to the administration of an anæsthetic is the position in which the patient is placed. The only proper position is with the head hanging down, the inhalation being commenced in the normal posture. This position ensures that no blood gets into the air-passages; thus the symptoms of suffocation and sickness are avoided. This, in view of the very smart bleeding which ensues, is important. Just as the existence of post-nasal growths causes paresis of the palate, for precisely

the same reason they cause stenosis of the nostrils, and, in addition, adhesions form far back, between the turbinate bones and the inner wall of the nostril. The first part of the operation is to dilate the nostrils; then the patient is gagged, and the finger is passed into the naso-pharynx, the nostril forcibly dilated with a sponge the size of a walnut, grasped in an ordinary dressing-forceps. The instrument is carried down the inferior meatus, up the middle meatus, and then out through the infra-meatus. That should be repeated twice. The blood at once begins to flow through the nose, and one is able to hear the breaking down of the adhesions. The next point is the removal of the growths, and for this purpose a great many instruments are used, varying from the natural finger-nail to the forceps. The finger-nail is distinctly bad, and the same may be said of the artificial nail. Then there is Gottstein's knife, which I never use because I do not think it is so serviceable as forceps. The knife is passed up, and then very forcibly passed downwards and backwards. I have seen a good many cases of recurrence of post-nasal growths, and in nearly all the instrument which had been used was Gottstein's knife. The cause of the failure to prevent recurrence is that the knife slips over the growths, and does not seem to get at the roots. The instrument which I approve of most is Lowenberg's forceps, or one of its modifications. Of course, in removing these growths one cannot see what he is doing, but the best way is to pass in one's finger ahead of the forceps, open them and grasp the growth, cut it off and take it out. Where there are several growths this has to be repeated a number of times.

The next point concerns the bleeding following the operation. Very often the haemorrhage in these cases is very smart, but, so far as the naso-pharynx is concerned, you can nearly always stop it by taking a sponge on the end of the forceps, passing it up, and pressing it forcibly into the naso-pharynx. If that does not answer, dissolve a teaspoonful of tannic acid in half a tumbler of water, dip the sponge in the solution, and pass it in. Having removed the growths and disposed of the cause of mouth-breathing and snoring, it is necessary to take steps to teach the child to breathe through the nose. A simple method is to make a cap to fit on top of the head and another to hold the chin, a broad piece of elastic being

stitched between them, which will act as a muscle to draw and keep the teeth together. If that fails, recourse must be had to putting court-plaster across the mouth. I must warn you against using lotions for the nose after operations until at least ten days have elapsed. One sees a good many cases where the use of lotion before that time has succeeded in driving some purulent matter into the ear, resulting in abscess in the tympanum.

I would next like to refer to enlarged tonsils, and, in doing so, to point out that in 75 to 80 per cent. of cases of enlarged tonsils there are also post-nasal growths. If the tonsils be removed and the post-nasal growths left, the symptoms will only be relieved, not cured, by the operation. Enlarged tonsils show a tendency to exist in particular families, just as do post-nasal growths. The exciting causes are the same—repeated acute attacks of tonsillitis always lead to some permanent enlargement of the tonsils. Acute tonsillitis may be either parenchymatous or follicular,—that is, the substance, or only the follicles, may be attacked. If only the follicles are attacked, there is some exudation accompanied by swelling, but not in so great a degree as in the parenchymatous form. For acute tonsillitis, the best treatment is to paint the tonsils with a 20 or 30 per cent. solution of cocaine, scarify freely, and give guaiacum lozenges for local use, and salicylate of soda internally. The symptoms which chronic tonsillitis gives rise to are very similar to those caused by post-nasal growths—obstruction to breathing, nasal voice, difficulty in swallowing, dryness of the throat, and there is the same general appearance as I described in connection with post-nasal growths.

With regard to the treatment of chronic tonsillitis, of course many methods are suggested, and very often when one sees cases for the first time, parents are very averse to operations, one reason given being that removal of the tonsils affects the sexual powers—quite an old wife's fable. The milder methods which such people desire tried first would consist in the application of astringent solutions; chloride of zinc, 30 grs. to the ounce is, I think, the best. Then there is puncture with the galvano-cautery, or burning with it. My own view of burning away large tonsils by means of the galvano-cautery is that it

is a dishonest means of making large sums of money, because months are occupied in the burning, whereas the whole tonsil could be removed in about ten seconds; moreover, I protest against a line of treatment which prolongs the suffering of the patient. I maintain that surgical removal of the tonsils is perfectly justifiable and legitimate, and produces the most satisfactory results. Just as strongly as I emphasised the necessity of giving a general anaesthetic when removing post-nasal growths, do I maintain the non-necessity of it when removing tonsils in adults. The tonsils can be very easily and freely cocaineised, and the duration of the operation does not exceed ten seconds. If a general anaesthetic be desirable, give nitrous oxide and oxygen. In the case of children, in view of the fact I have mentioned, that in 80 per cent. of these cases of enlarged tonsils there are post-nasal growths as well, the general anaesthetic will enable both to be removed at the same time. In removing tonsils in adults, first paint the bodies freely with a 20 per cent. solution of cocaine, allow a few minutes to elapse, then operate. When a general anaesthetic is used the patient should be placed with the head down, as previously described, and the tonsils should be removed before the post-nasal growths are attacked or the nose dilated. If the nose be dilated first, a certain amount of blood trickles into the pharynx and prevents the operator seeing his work. Of course a gag should be used with a general anaesthetic. The best instrument for removing tonsils is the guillotine I show you, which is known as Mackenzie's modification of Physick's guillotine; with due caution this never fails. If the operator is not ambidextrous, he must have someone to assist in pressing in the tonsils. First place the thumb outside the tonsil and the fingers on the side of the cheek; then introduce the tonsillotome horizontally over the thumb in the way I show you, then lateralise it, then press back the ring behind the tonsil. With this as a fulcrum draw the handle of the tonsillotome from the centre line against the corner of the mouth, a little pressure will cause the patient to gag and attempt to swallow; this is the time to cut. Rapidly withdraw the instrument, and cut out the other tonsil without loss of time, as the patient will very likely want to spit or take a "few breaths" before you attack the other, and in some

cases where this is allowed you may not succeed in getting the mouth open again. I have never yet seen a serious case of bleeding after this operation but on one occasion, and then a tonsillar artery did not begin to bleed until hours after the operation. Fortunately the patient was within reach, and the haemorrhage was stopped by taking hold of the bleeding point and twisting it. For general bleeding it is quite sufficient to use cold water, or a tumbler of cold water into which has been put a teaspoonful of tannic acid. The patient should be directed to fill his mouth with this, then hold back his head, and afterwards spit the liquid out. The pain following the operation is sometimes considerable; it is then desirable to get the patient to use marshmallow lozenges. In any acute condition of the throat, especially after a surgical wound, marshmallow forms a gelatinous film on the surface and relieves pain.

I next want to speak of follicular pharyngitis, or what is commonly known as "clergyman's sore throat." You are aware that the surface of the mucous membrane in the pharynx is studded with small glands secreting mucus to lubricate the throat. From one of the causes which I will mention, these glands become inflamed, the secretion becomes thickened or inspissated, the mouth of the gland is blocked, and on coming to examine the throat one finds a number of small, red, fleshy-looking elevations, which constitute the follicles of follicular pharyngitis. It is due to any cause which brings on chronic irritation in or chronic discharge from the naso-pharynx, or from the pharynx itself. Therefore, in nearly all cases of enlarged tonsils, or naso-pharyngeal adenoma, there is follicular pharyngitis. The disease, of course, may exist without those other conditions, and is found in persons who use their voices unskillfully, and those who are accustomed to talk loudly in the open air. The symptoms which follicular pharyngitis gives rise to are dryness of the throat, and a constant desire to clear it. A parson or public speaker will usually say he is able to begin his sermon or speech all right, but when he gets to the "tenth head" his voice begins to fail, and he ends up almost voiceless, and with a very unpleasant sensation of pain in his throat, the pain of muscular exhaustion. On examination, one probably finds that he has not only follicular pharyngitis, but some of its sequelæ; and,

especially if this condition be complicated by pharyngeal trouble, there is a tendency to congestion of the mucous membrane of the larynx and vocal cords. These troubles always travel downwards. If there be simply a post-nasal catarrh, a lotion such as I have mentioned sniffed up the nose, or inhalations of oxygen, give great relief. Steam inhalations are very useful; but they are more serviceable for the treatment of pharyngeal than for naso-pharyngeal trouble. Generally, one begins with mild methods, such as astringent paints, carbolic acid lozenges (which are useful in relieving dryness of the fauces), steam inhalations, or the vapour of benzoin. For the radical treatment you must use the galvano-cautery. A few years ago it was the custom to burn these enlarged follicles with one of the caustic pastes; but that treatment has long since been abandoned in favour of the galvano wire. The objections to the paste are that when the wood carrying the paste is being introduced, the patient may gag and the tongue gets burnt; secondly, the paste may be applied to the wrong place; and thirdly, if you put in paste just sufficient to burn one follicle an eschar results, much larger than is necessary. The advantage of the cautery wire is that it is not brought into action until it is on the spot you desire to burn, and you can choose a point corresponding in fineness to the area to be burnt. It is advisable to cauterise only one side of the pharynx at a time, waiting about ten days before attempting the other side. If both sides were done at once the patient would have a very sore throat; besides which it is better to leave them one side for swallowing. After the follicles have been burned, the mucous membrane can be treated, and this can be efficiently done by the use of paints and lozenges. I do not recommend gargles, because their application is limited to thirty or forty seconds at a time, whereas lozenges produce a continuous effect. Inhalations may be afterwards used.

Of course it is impossible, in the time at our disposal here, to give you much practical instruction in carrying out the various methods of treatment which have been suggested, but I will be happy if any of the gentlemen present will attend on Fridays, about 1.30, when a better opportunity will be afforded them.

[A number of cases were then examined and demonstrated.]

THERAPEUTICAL NOTES, &c.

A Laryngeal Diaphragm.—Such a membranous formation was observed by P. Bergengrun in the larynx of a woman, 41 years of age, who for fifteen years had had some difficulty in respiration, without any disturbance of speech. The dyspnoea, at first slight, had increased gradually, until it became so intense as to cause the woman to seek medical assistance. The functional symptoms were as follows:—Inspiratory depression of the sub-clavicular fossæ and the intercostal spaces; noisy, strident inspiration and expiration; feeble, but not hoarse voice. A rapid and slightly irregular pulse was noted; the general condition was not very good, but not bad; the heart, lungs, nose, and pharynx were normal. The larynx was small, the form, colour, and movements of its different parts being normal. A little below the glottic space was a yellowish-grey membrane, shining like a tendon, and completely obstructing the trachea. In the neighbourhood of the left arytenoid a small opening as large as the head of a pin was seen. It was the only orifice by which the air could pass. The membrane was incised as far as the posterior commissure on one side and almost as far as the anterior commissure on the other. There was but an insignificant haemorrhage, and respiration immediately became noiseless. Four days later the introduction of Schrötter's ebonite tubes was begun, and on the tenth day No. 7 of the series could be passed and the voice and respiration had become entirely normal. The patient then left the clinic, and when seen two months later there remained of the diaphragm only two small subglottic projections at the anterior commissure.

The author regards it as remarkable, from a physiological standpoint, that no disturbance of the voice should have accompanied the almost complete cutting off of the air. Contrary to theoretical ideas, it would appear from this case that a very small quantity of air suffices for the production of normal sounds. It is probable that a greater force of the expiratory air-current made up for the small quantity; the larynx and the supra-laryngeal portions of the respiratory passages were acted upon with more force, and the value

of the pharynx, nose, and sinuses as resonance cavities thus increased.

Arch. f. Laryng. u. Rhinol.

A Possible Effect of Antitoxin.

By E. CROSBY CHAMBERLIN, M.D., New York.

Without discussing the value of diphtheria antitoxin, when early administered, I wish to relate a case which is of some interest. A young woman, aged 22, has been afflicted with a chronic diarrhoea for the past four years. In December I attended her for a gastritis, which after a few washings of the stomach passed away. Two months later diphtheria developed; being pharyngeal at first, it extended in all directions until two days later, when an otitis media with a copious discharge was developed. This day the report from the Board of Health was received confirming the diagnosis. She had been using for two days corrosive sublimate, both internally and as a gargle. The discharge from her ears by this time was so abundant she could not sleep, it flooding out of the external meatus over the face through absorbent cotton and everything. I at once gave her two grains of antitoxin. The next day the discharge was reduced at least one-half. Later she received another injection of antitoxin, and the following day not only had the discharge entirely disappeared, but the diarrhoea was reduced from five or ten stools daily to one or two stools of a normal solid consistency.

That was two months ago, and at present she has not had a return of the distressing diarrhoea.

Medical Record.

Hæmoptysis.—Dr. Desplats, of Lille, advises, when the haemorrhage continues in spite of the first measures employed, and when a preparation of ipecac is not at hand or it is not desirable to use it, to take half a glass of water, quickly dissolve it in a tablespoonful of ordinary salt, and give it as a single drink. The effect is sometimes instantaneous. If this does not succeed, ligature of the four members should be the last resort, the ligatures being loosened one by one, giving time for a clot to form, and gradually so as not to increase the arterial tension too rapidly.

Journ. de Méd. et de Chir. Prat., June 10, 1896.

INDEX TO VOLUME VIII.

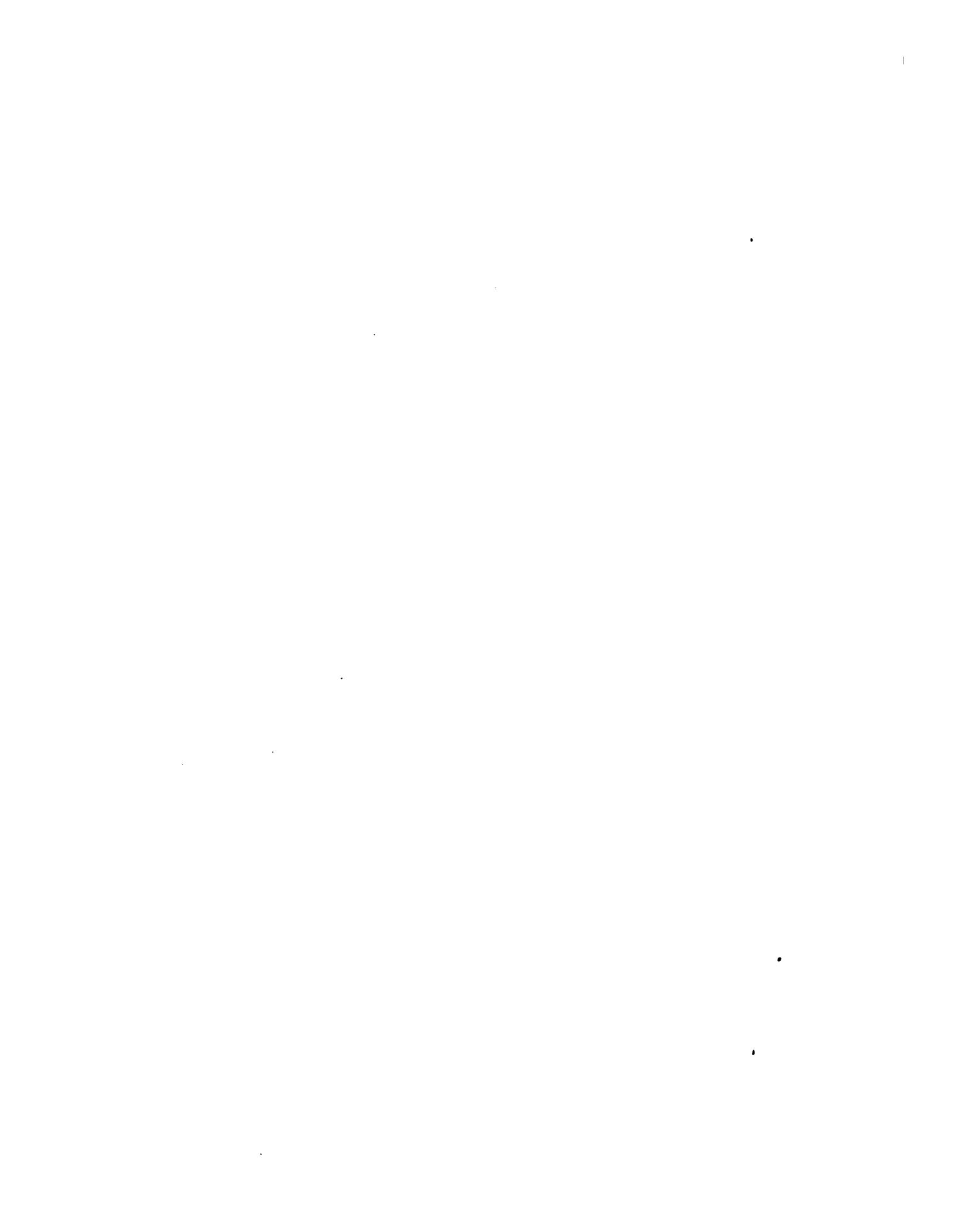
A	PAGE	PAGE	PAGE
Abdominal tumour	32	Arthritis, rheumatoid, treatment of	17
" simulating cirrhosis	83	Artificial feeding of infants, formula	302
" wound, mode of closure of	195	Ascites	82
Abscesses, cerebellar	345	Ataxia in Friedreich's disease	119
" cerebral	344	Atrophy, diagnosis of	296
" intra-cranial	344	kinds of	294
" multiple tubercular sub-cutaneous	231	muscular	294
Acne	356	order of spread of	296
" causes of	356	v. poliomyelitis	299
Acromegaly, therapy of	48	B	
Addison's disease	189	Bakarewitsch treatment of varicose ulcers	384
Adenoid vegetations	392	Barker, Mr. A. E., on weak scars and herniae following abdominal sections	193
" accompanying symptoms of	278	Bartholinitis, treatment of	334
" causes of	277	Beever, Dr., on paralysis of the soft palate	376
" instruments for removing	282	Bladder, new growths in the	68
" naso-pharyngeal	276	Bleorrhoea, dermatol in	332
" prognosis when not removed	279	Blood, tests for	67
" treatment of	334	Bougies	205
" types of	277	Brain complications in ear disease	344
Adenoma sebaceum	208	Bright's disease, chronic	273
Alcohol as a cooling agent	42	" diagnosis of	273
Alcoholic peripheral neuritis	18	" importance of rest in	275
Alcoholism, formula for	176	" symptoms of	274
" with suggestions as to treatment	176	" treatment of	275
Alopecia, formula for	367	Bröncho-pneumonia	243
" treatment of	367	" treatment of	246
Amputation of breast	109	Burns, new treatment for	303
" ulcerated leg	38	C	
Anæmia of Hodgkin's disease independent of leukocytosis	35	Calculus, diagnosis of, by new photography	348
Anastomosis, intestinal, some methods of performing	337	Cancer of liver	361
" with Murphy button	319	" of rectum	356
Aneurysm	192	" of tongue	289, 355
" cases of	95	" after-treatment in	293
" which simulate	139	" cases of unsuitable	
" intra-thoracic	136	" for operation	292
" signs of	138	" causes of	289
" treatment of	140	" diagnosis of	290
Anorexia nervosa	18	" duration of life in	290
Antitoxin, possible effect of	408	" Kocher's operation	
Antral suppuration following invasion by dental cyst	394	for	292
Antrectomy for chronic middle ear disease and facial paralysis	14	mode of commencement	
Aphonia, hysterical	107	of	289
Apoplexy, importance of absolute rest in	47	Cancerous tubercle v. cirrhosis	83
Appendicitis, a study of	310	Carbuncle, surgical treatment of	351
" classification of	15	Carcinoma of oesophagus	109
" diagnosis of	15	Cardiac compensation	130
" distinction from typhlitis	16	Caries, treatment of	344
" evolution of	328	Cartilages, loose	211
" extra-peritoneal complications of	331	" semilunar, displacement of	237
" onset of	327	Castration for hypertrophied prostate	10
" pathogenesis of	311	Cataract, operation for	93
Arsenical poisoning	372	Cavafy, Dr., on enteric fever	221
Arthritis, gonorrhœal	10	Celluloid mull bandage	384
" rheumatoid	17	Cerebral tumour	369
		" symptoms of	371
		Cerumen, removal of, from auditory canal	255
		Cervical torsion	375
		Chancre, hard infecting, of eyelid	173
		Cheiro-pompholyx of eyelid	208
		Chloroform commission, 'Lancet' report on	267, 283
		Chorea, rheumatic, nature of	20
		" traceable to school pressure	20
		Chronic Bright's disease	273
		" diagnosis of	273
		" symptoms of	274
		" treatment of	275
		" glandular disease of the nose and naso-pharynx	403
		Cirrhosis, hypertrophic	84
		" of liver, a cause of dropsy	82
		" and abdominal section	190
		" causes of	81
		" definition of	81
		" diagnosis of	81
		" in children	305
		" causes of	307
		" diagnosis of	309
		" forms of	305
		" mordid anatomy of	305
		" prognosis of	306, 309
		" symptoms of	309
		" treatment of	310
		" pleuritic effusion in	192
		Clark, Mr., a note from the clinic	302
		Cleft palate, best time for operating on	11
		" mode of operating on	11
		Clinical cases at the museum	24, 206,
		" 230, 271, 277, 333, 353	
		" N.W. London Society	
		" 30, 94, 172	
		" notes	100, 151, 188, 232, 389
		Cold ablutions, when indicated	43
		" in delirium tremens	221
		Collateral circulation in portal obstruction	85
		Collins, Mr. E. T., on orbital and ocular tumours	5
		Colotomy	261
		Colour vision and colour blindness (review)	303
		Complications of middle ear suppuration	340
		Consultations at St. Bartholomew's Hospital	38, 63, 78, 109, 191
		" at St. George's Hospital	237
		Corneal inflammation	262
		Coryza, formula for	40
		Cough, tonsillar	253
		Craniotomy for idiocy	254
		Creasote in phthisis	190
		Croupous pneumonia	241
		" acute treatment of	398
		Curettage for puerperal fever	16
		Current from the main (review)	303
		Cysts, dermoid	5
		" hydatid	5
		" implantation	5
		" irrigation and drainage after removal of	7
		" removal of	5

D	PAGE	PAGE	PAGE
Death, apparent, in newly born, treatment of	396	Exostosis	390
Deformities associated with peripheral neuritis	39	" multiple	333
Delirium tremens, cold baths in	221	Eye, researches into anatomy and pathology of the (review)	144
Derangement of knee-joint	209	F	
Dermatitis herpetiformis	230	Fibula, sarcoma of	240
" traumatic	207	Fistula, urinary	260
Dermatological Congress, notes at	247	Flatulence a cause of hernia	321
Diabetes mellitus, treatment of	19	Friedreich's disease	118
Diagnosis, problems in practical	256	" aetiology of	119
Dietary in enteric fever	43	" diagnosis from disseminated sclerosis	122
Dieulafoy, Prof., on a study of appendicitis	310, 327	" diagnosis from true tabes	122
Diphtheria, absence of bacillus not conclusive	1	" morbid anatomy of	119
" anuria with vomiting, a fatal sign in	2	" treatment of	122
" bad after-effects of antitoxin	2	Functional paralysis	102
" beneficial effects of antitoxin	2	G	
" comparative mortality in diagnosis in absence of bacillus	3	Gall-stones	112
" mode of administering antitoxin in	1	Gardner, Mr., on nitrous oxide and gas for removal of adenoid growths	301
" nephritis a rare complication in	2	Gastric complications in post-diphtheritic paralysis	188
" paralysis after	4	Glioma of retina, not always fatal	9
" persistence of bacillus after disappearance of membrane	4	" recognition of	9
" prompt injection important	4	Gonorrhœa, prevention of	384
" significance of streptococcus in	1	Gonorrhœal rheumatism	190
Disease of middle ear	391	Gould, Mr., notes from the clinic	257
Diseases of rectum and anus (review)	96	Gout, aetiology of	354
Dislocation of elbow	78	" diet as a factor in	355
Diuretin in morbus cordis	189	" forms of	354
Drains after abdominal sections	194	" in relation to digestive system	202
Duboisin, intoxication from use of	320	" lymphatic	202
Duckworth, Sir Dyce, clinical notes	17	" respiratory	202
Dwarf, short-limbed	333	" influence of nervous system in production of	198
E		" probable causes of	197
Ear discharge, treatment of	340	" relation of defective metabolism to	197
" disease, diagnosis of	123	" v. rheumatism	355
" instruments used for	125	Gowers, Dr., on slight multiple neuritis	21
" symptoms of	124	Graft for ulcerated leg	39
" examination of	123	Green, Dr., on chronic Bright's disease	273
Eclampsia, modern treatment of	400	Guthrie, Dr., on unilateral paralysis of the ocular sympathetic	225
Ectopic testis	97	H	
" operation for	97	Habershon, Dr., on mitral stenosis	129
Embolism, cortical, arising from mitral stenosis	19	Hadley, Dr., on cerebral tumour and arsenical poisoning	369
Emergencies, treatment of some medical	41	Haematuria, diagnostic significance of	67
Endemic haematuria	73	" endemic	73
Endocarditis, ulcerative	20	" in vesical tumour	48
Enteric fever	43, 221	" paroxysmal	71
" complications of	223	" renal	70
" course of	222	" some causes of	65
" grey powder in	44	" treatment of	69
" onset of	221	Hæmoptysis	46, 408
" oysters, a cause of	222	" formula for	62
" relapse in	44, 223	" in phthisis	47
" treatment of	224	Hæmorrhage, accidental	28
Epididymitis, tubercular disease of	110	" guarded prognosis necessary in	30
Epistaxis in chronic Bright's disease	275	" in brain surgery	398
Epithelioma of jaw	64	" prostatic	68
" of tongue	87	" treatment of	29
" on cheek	271	" urethral	68
Eruption due to bromide of potassium	272	" vesical	68
" undiagnosed	230	Hæmorrhoidal flux	304
Erythematous eruption	26	Harrison, Mr., on stricture of urethra	204
		Hawthorne, Dr., on cancer of liver	361
		Headache in chronic Bright's disease	275
		" periodic, formula for	108
		" persistent	19
		Hearing power, test of	126
		Heath, Mr., on tetanus	177
		Hemiplegia after sunstroke	143
		" and pregnancy	191
		" haemorrhagic	103
		" infantile	101
		" mitral stenosis in	19, 30
		Hernia, double congenital	238
		" femoral, rarity of	322
		" flatulence a cause of	321
		" in childhood	321
		indications for operating in	324
		" inguinal	322, 325
		" treatment of	323
		" trusses for	323
		" v. retained testis	323
		" its relation to phimosis	321
		" strangulation of	322
		" umbilical	99, 322
		" unsuitable case for radical cure	134
		" varieties of	321
		Hernia following abdominal sections	193
		Hernial trusses, application of	299
		Hiccup cured by traction of tongue	352
		Hip-joint disease, mode of operating for	163
		" operation in children	161
		Hodgkin's disease	33
		" autopsy on a case of	37
		" diagnosis of	33
		" differentiation from syphilitic glands	37
		" distinction from leucocythaemia	34, 37
		" treatment of	37
		Hopkins, Mr., on surgical sequelæ of chronic nerve disease	113
		Hutchinson, Mr. Jonathan, clinical cases	24, 206, 230, 277, 333, 353
		Hydrocephalus, treatment of	13
		Hyperpyrexia, ice packs and graduated bath in	42
		" use of drugs in	42
		Hypertrophic morphea	79
		Hypnotism, its practical uses in public clinics	380
		I	
		Icterus infectious	398
		Idiocy, craniotomy for	254
		Incontinence of urine	128
		Infantile hemiplegia	100
		Infants, artificial feeding of, formula for	302
		Influenza	317
		Internal derangement of knee-joint	214
		" diagnosis of	216
		" prognosis in	217
		Intoxication from use of duboisin	320
		Iridectomy	92
		" cases suitable for	92
		Iris, frequent seat of cystic growths of eye	7
		Ivory exostosis	6
		J	
		Jacobson, Mr., epithelioma of tongue	87
		Jaundice due to impacted gall-stone	60
		" treated by mercury	207
		Johnson, Dr., the application of hernial trusses	299
		" Mr. Raymond, on hernia in childhood	321

K	PAGE		PAGE	PAGE	
Keloid in scars of superficial burns	25	Meningitis, method of operating for	52	Paralysis of ocular sympathetic, symptoms of	225
Keratosis	250	" not always tuberculous	49	" of soft palate	376
" with hyperidrosis	27	" prognosis of	51	" " causes of	378
" treatment of	27	" surgical aspects of	49	" " prognosis in	379
Knee-jerk, reinforcement test of	21	" symptoms of	50	" post-diphtheritic	17, 188
		" treatment of	58	" treatment of	17
L		" trephining of skull in	55		
Lane, Mr. Arbuthnot, clinical notes	10	Metatarsalgia	349	Paraplegia, ataxic	103
Laryngeal diaphragm	408	Middle ear suppuration, some complications of	340	Paroxysmal haematuria	71
" perichondritis	187	Mitral stenosis	129	Perforating ulcer, diagnosis and treatment of	302
Larynx, gumma in the	185	" diagnosis of	133	Periarthritis, acute temporal	172
" syphilis in the	180	" " v. mitral regurgitation	131	Perichondritis, laryngeal	187
Lees, Dr., on appendicitis	14	Morbus cordis, diuretin in	189	Perihepatitis a cause of portal obstruction	82
Leucocytæmia	59	Morphine disease, diagnosis of	350	Peripheral neuritis	39, 172
" v. leukocytosis	37	" tests for	351	Peritonitis, acute	41
Leucorrhœa, formula for	27	Morphea herpetiformis (nigra)	24	" following perforation of stomach or intestine	41
Liver, cancer of the	361	" due to causes acting through nervous system	24	" opium and hot fomentations in	41
" contraction of, in heart disease	83	Morton, Mr., on the surgical treatment of tuberculous disease of bladder	157	Phimosis in relation to hernia	321
" syphilitic disease of	84	Muscular atrophy	294	Phonographic Record of Clinical Teaching (review)	47, 144, 256
Lockwood, Mr., on an unsuitable case of hernia for radical cure	134	" diagnosis of	296	Phthisis, crease in	190
" on a suitable case of hernia for radical cure	401	" kinds of	294	Pick, Mr., on operative treatment of hip-joint disease in children	161
Locomotor ataxia	141	" order of spread of	296	Pigmentation of lips of mouth	231
Loose body in knee-joint	63	" v. poliomyelitis	299	Pinard, Professor, on treatment of apparent death in the newly born	396
" cartilages in joints	209	Myxœdematos patient, tuberculous ulcers in	38	Pityriasis rosea simulating syphilis	232
" " analysis of cases of	211			Pleuritic effusion in hepatic cirrhosis	192
" " diagnosis of	212	N		Pleuro-pneumonia	17
" " treatment of	213	Nævi of choroid	9	Pneumonia, acute	45
Luff, Dr., clinical notes	59	Nævus suitable for operation	111	" venesection in	45
Lumbago, formula for	100	Narcosis, improved method of	384	Pneumonias in children and their sequælae	241
Lupus, syphilitic	272	Necrosis and epiphysitis v. tubercular epiphysitis	98	" diagnosis of	242
" vulgaris	25, 230	" of femur	11	" prognosis of	243
Lymphadenoma, aetiology of	233	Nerve disease, sequælae of chronic	113	" treatment of	243
" autopsy of case of	37	Neuritis, alcoholic peripheral	18	" v. tuberculosis	244
" diagnosis of	33	" multiple peripheral	23	Pollard, Mr., on ectopia testis	97
" differentiation from leukocytæmia	34, 37	" following injury	23	Polyp of frontal sinus	28
" prognosis of	234	" its wide range of causation	24	" operation for	28
" treatment of	37	" slight	21	Polypi, auræ	341
Lymphatic conjunctivitis	262	" peripheral	172	Polysarcia	18
" " causation	264	Nitrous oxide and oxygen for removal of adenoid growths	301	Portal obstruction, collateral circulation in	85
" " treatment	264	Notes at the Oxygen Hospital	252	Power, Mr., on meningitis in its surgical aspects	49
		Nux vomica in diseases of children	349	Procidentia uteri	145
M				" causes of	146
MacCormac, Sir Wm., on cancer of the tongue	289			" instruments used for	149
" " on haematuria	385	O		" symptoms of	147
" " on internal derangement of knee-joint and loose cartilages in joints	289	Ocular sympathetic, paralysis of	225	" treatment of	148
" " on diseases of the tongue	65	Onychia in an infant	26	Prostate, hypertrophied, radical treatment of	333
Mackenzie, Dr., on diagnosis of cirrhosis of liver	81	Optic neuritis following ear disease	345	Prostatic haemorrhage	68
McKerron, Dr., accidental haemorrhage	28	Orbital contents, method of excision	6	Prurigo	250
Malformation of both hands	63	" growths, diagnosis from dis tended frontal sinus	6	Pruritus, senile	349
Martin, Dr., on treatment of diphtheria by antitoxin serum	1	" method of examining for	6	" vulvar and anal, treatment of	364
Mastoid disease	11, 342	O		Psoriasis	32, 206
" " operative indications	343	Ocular sympathetic, paralysis of	225	" of almost universal extent	26
Melæna, theory to account for	128	Onychia in an infant	26	Public Health Laboratory Work (review)	272
Meningitis, diagnosis from typhoid fever and pneumonia	50	Optic neuritis following ear disease	345	Puerperal fever, curettage in	16
" following ear disease	346	Orbital contents, method of excision	6	" sepsis, indications for operating in	318
" forms of	49	" growths, diagnosis from dis tended frontal sinus	6	Pulmonary tuberculosis, types of	168
" histological results of	56	" method of examining for	6	Purpura, prognosis in	235
" measurement of pressure of cerebro-spinal fluid in	55	O		" treatment of	237
		Paralysis, facial	103	Pyæmia following ear disease	348
		" functional	102	Pyelitis	62
		" of ocular sympathetic	225	Pyosalpinx	175
		" " " action of mydriatics in	226	Q	
				Quadriceps extensor, rupture of	368

R	PAGE	S	PAGE	T	PAGE	U	PAGE	V	PAGE	W	PAGE
Rachitis and its relation to dampness	398	Syphilis at eleven years of age	21	Tumours in head of infant	94	Ulcer, crateriform	271	Vaccinia, diffuse, general	173	Warty growth of tongue	89
Raynaud's disease	31	" extra-genital, prognosis of	249	" ocular, classification of	5	" perforating	113, 302	Waterhouse, Dr., on adenoid vegetations in naso-pharynx	276	Waterhouse, Dr., on the practical uses of hypnotism in public clinics	380
Rectal ulcers and the electro-cautery	317	" malignant	248	" of optic nerve, rarity of	10	" rodent, on nose	27	Wethered, Dr., on types of pulmonary tuberculosis	168	Whistler, Dr., on syphilis as it affects the larynx	180
Rectum, cancer of the	356	" pathology of	185	" symptoms of	10	" varicose, cause of	12	Whooping-cough	217	Whooping-cough	46
" diagnosis of	356	" signs of	182	Turner, Mr., on antral suppuration following invasion by a dental cyst	394	Worms	271	Wilkin, Dr. W. P., on the practical uses of hypnotism in public clinics	380		
" operation for	359	" tertiary	95	" of thigh	174	Xanthelasma palpebrarum	353	" Mr. G. C., on polyp of right frontal sinus	28		
Renal dropsy	84	" treatment of	187	Turner, Mr., on antral suppuration following invasion by a dental cyst	394	Xantheloma multiplex	533	William's, Dr. Theodore, on arrest of pulmonary tuberculosi	74		
" haematuria	70	Syphilitic contraction of liver	81	Typhoid fever, feeding in	189	Xanthomatous patient	38				
Resection, conditions requiring, and instruments used	337	disease of liver	84	Umbilical hernia	99	Umbilical hernia	99				
Retinal growths	9	lupus	272	Urethra, stricture of	204	Urethral haemorrhage	68				
Rheumatism, acute, diagnosis of	17	re-infection	247	Uric acid, comparative value of remedies for	255	Urethral fistula	260				
gonorrhoeal	190	syco-s-lupus	207	V		Venesection in pneumonia	45				
Rickets, cause and treatment of	12	tongue becoming epitheliomatous	80	Vaccinia, diffuse, general	173	Verruca plana	271				
" pathology and treatment of	319	ulceration of tongue	387	Varicocele	192	Varicose haemorrhage	68				
Rodent ulcer on nose	27	Syringomyelia, condition of deep reflexes in	298	Varicose saphena vein, ligature preferable to excision	13	" haemorrhoids	70				
Rolleston, Dr., clinical notes on cirrhosis of liver in children	141, 305	" diagnosis of	297	" ulcers, Bakarewitsch treatment of	384	" diagnosis of	70				
Routh, Dr., on procidentia uteri	145	" diseases which produce	298	" cause of	12	Vocal cords, impaired movements of	104				
Rupture of bladder, diagnosis of	70	" distribution of	297	" " causes	106	" " symptoms	105				
S		" phenomena of	296	" " treatment	107	Vomiting in chronic Bright's disease	275				
Sarcoma, liability to, of blind and shrunken eyes	8	" treatment of	299	W							
" of alveolus	10	Taylor, Dr. F., on an unusual case of Hodgkin's disease	33	Warty growth of tongue	89						
" of ciliary body and choroid	8	" Dr. Seymour, on treatment of some medical emergencies	41	Waterhouse, Dr., on adenoid vegetations in naso-pharynx	276						
" of fibula	240	Tetanus, cause of	178	Wethered, Dr., on types of pulmonary tuberculosis	168						
" of male breast	89	" definition of	177	Whistler, Dr., on syphilis as it affects the larynx	180						
" orbital	90	" treatment of	179	Whooping-cough	217						
" v. inflamed veins	6	" v. hydrophobia	178	" morphia in	46						
Scars, treatment of	116	Thrombosis of lateral sinus following ear disease	347	Wilkin, Dr. W. P., on the practical uses of hypnotism in public clinics	380						
" weak, conditions determining formation of	193	" of portal vein	83	" Mr. G. C., on polyp of right frontal sinus	28						
" following abdominal sections	193	" treatment of	347	Williams, Dr. Theodore, on arrest of pulmonary tuberculosi	74						
Sclerosis of mitral valve	28	Tinnitus	253	X							
Scrotal tumour	374	Tongue, cancer of, after-treatment in 293	393	Xanthelasma palpebrarum	353						
Sepsis, puerperal, operation for	318	" cases unsuitable for	293	Xantheloma multiplex	533						
" treatment by intra-venous serum injections	253	" operation	292								
Sequelæ of chronic nerve disease	113	" causes of	289								
Sheild, Mr., notes from the clinic	380	" diagnosis of	290								
Shock in acute peritonitis	41	" duration of life in	290								
Shorthand in medicine	48	" diseases of the	389								
Skin grafting, material for	335	" epithelioma of	87								
Sleeplessness	366	" new growth of the	388								
Smith, Dr. F. J., clinical notes	188	" pre-cancerous ulceration of	88								
Snake poisoning, treatment of	336	" tuberculous ulceration of	387								
Soft chancre, formula for	128	" warty growth of the	89								
" palate, paralysis of	376	" tumours of the	389								
Spicer, Mr., on lymphatic conjunctivitis	262	" Kocher's operation for	292								
Stewart, Mr., on examination of the ear	123	Tonsillitis, acute, formula for	368								
" on some complications of middle ear suppuration	340	Tooth, Dr., clinical notes	100								
Stoker, Dr., on impaired movements of vocal cords	104	Torticollis, congenital, treatment of	218								
" on chronic glandular disease of the nose and naso-pharynx	403	Trichloracetic acid as a cure for chronic otorrhoea	352								
Stomach, drainage after incisions in	42	Trusses, hernial, application of	299								
Stricture	257	" essential parts of	300								
" importance of rest in	258	Tubercle a cause of ear disease	124								
" of urethra, and instruments used for	204	Tubercular abscesses of foot	91								
Suppurative folliculitis	25	" mastoid disease	389								
Supra-pubic puncture, the technique of	367	" peritonitis	83								
Suture of intestine, best material for	337	Tuberculosis, clinical types of	168, 249								
Swelling of foot due to improper boots	12	" diagnosis of, from morphology of blood	399								
Syphilis a cause of ear disease	124	" pulmonary, arrest of	74								
" duration of period of contagion of	247	" changes found in	75								
" as it affects the larynx	180	" ulceration of tongue	387								
		Tumour, abdominal	32								
		" congenital, of sterno-mastoid	218								
		" treatment of	218								





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19 of
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